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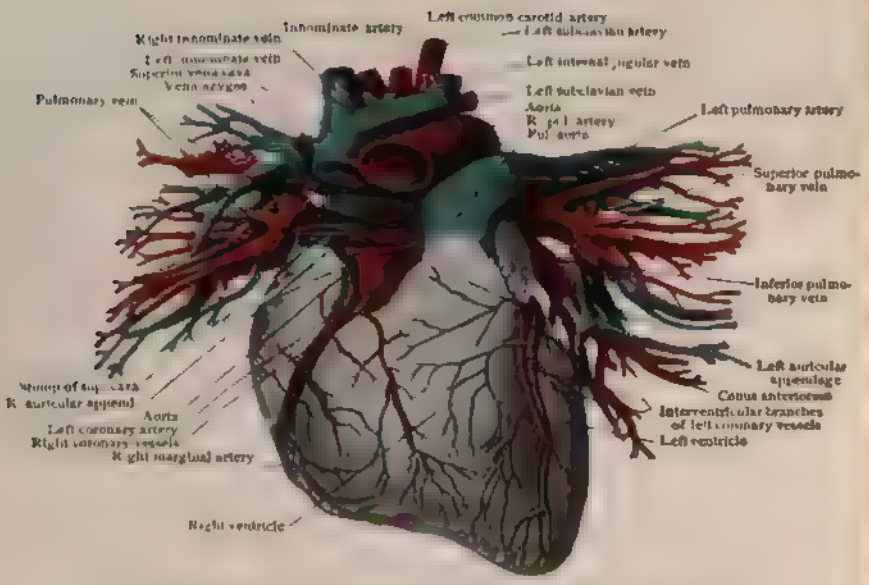

FIG. 1.—The heart and great vessels, viewed from the front. (After Piersol.)

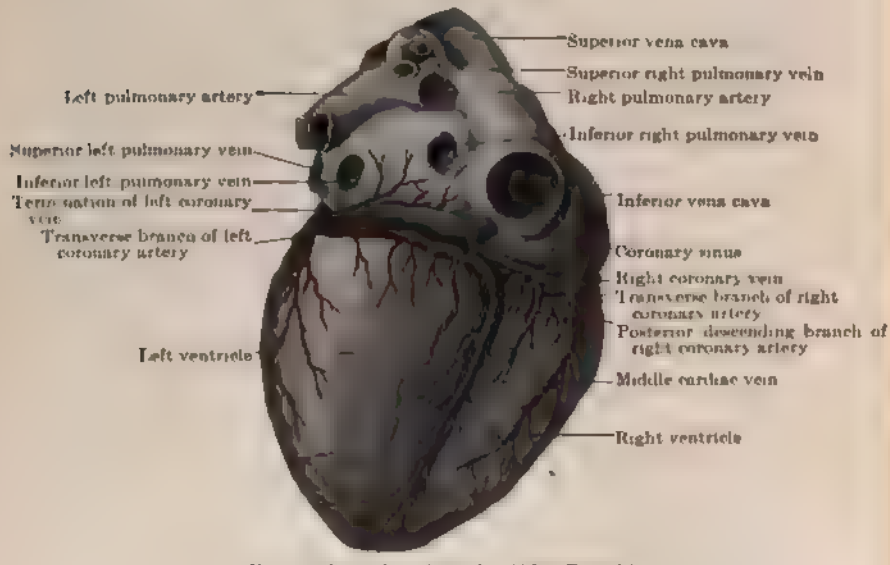

FIG. 2.—Same, from behind. (After Piersol.)

DISEASES

of the

HEART AND AORTA

BY

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WITH AN INTRODUCTORY NOTE

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329 ILLUSTRATIONS BY THE AUTHOR

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1910

TO

MY FATHER

Joseph Oakland Hirschfelder, M.D.

Professor of Clinical Medicine, Leland Stanford Junior University

AND TO

Lewellys F. Barker, M.D., LL.D.

Professor of Medicine, Johns Hopkins University

CHIEFS OF THE CLINICS IN WHICH THE WORK WAS DONE; WHO
HAVE TAUGHT ME BY PRECEPT AND EXAMPLE HOW
SCIENCE, ART, AND HUMANITY SHOULD
BE WOVEN INTO PRACTICE
OF MEDICINE

THIS BOOK IS AFFECTIONATELY DEDICATED

27593

INTRODUCTORY NOTE

THE researches in the great field of inner medicine have so multiplied in recent years that it has become highly desirable that we should have from time to time, in addition to the summaries of progress contained in the general text-books on practice, monographs which picture more completely the status of our knowledge in the several special divisions of the subject. In diseases of the circulatory system new methods of study have led to the discovery of many new facts, and a great many workers have been attracted during the last twenty years to this domain of cardiovascular inquiry.

In the medical clinic at the Johns Hopkins Hospital, Dr. Hirschfelder has during the past few years occupied himself especially with such studies. The present volume is an attempt to epitomize the actual condition of the subject at the present time, as viewed from the standpoint of an active investigator of extensive first-hand experience who has also a wide acquaintance with the literature of the physiology and pathology of the circulatory apparatus.

The clearness and brevity of the presentation and the excellent arrangement of the material will, I am sure, appeal to students and practitioners of medicine. It is no easy matter adequately to combine the most recent results of anatomical, physiological, pathological, and clinical studies in a form which will satisfy the critical demands of the scientific investigator and at the same time be useful as a guide to the every-day practitioner. Especial attention has been paid in the volume to the practical facts of diagnosis and treatment; in the more theoretical portions there will be found evidence of careful, critical sifting, and an appreciation of the distinction between what is essential and what non-essential for the more general reader.

The bibliographic references make no attempt at completeness, but have been chosen with the idea in mind of permitting those who desire to do so to consult the most important, and especially the more recent, treatises, monographs, and original articles which deal with the various matters discussed.

A notable feature of Dr. Hirschfelder's book is the liberality of illustrations; the majority of the figures are made from original drawings and tracings and are in pleasing contrast with the time-worn figures which pass from compilation to compilation.

LEWELLYS F. BARKER.

BALTIMORE, May 12, 1910.

PREFACE

In the preparation of this book it has been the writer's aim to present side by side the phenomena observed at the bedside and the facts learned in the laboratory in order to show how each supplements the other in teaching us how to observe the patient and to direct the treatment. Many of the results obtained in the laboratory have not yet attained practical importance because they have been scattered through the literature and have not reached the eye of the clinician; but wherever the clinicians have looked to the laboratory or laboratory workers have looked to the clinic for verification or application of their theories the great pillars of progress have been raised. In accordance with this idea the clinical presentation in each chapter is preceded by an introductory section dealing with the experimental pathology and more fundamental principles of the subject, which has been used as a basis for frequent reference in the clinical discussions.

The trend of clinical observation during the past two decades has been toward more accurate study of disturbances of function and toward the introduction of mechanical methods for their observation, methods of precision which tend to supplement or supplant the older and simpler methods of physical diagnosis. Chief among these may be mentioned the study of blood-pressure, the graphic studies upon alterations in cardiac rhythm by means of the venous pulse, the outlining of the heart and vessels by means of the X-ray, and the phonographic recording of the heart sounds. Each of these subjects has been reviewed with special reference to the general principles upon which the method is based, in order to point out its applicability, its limitations, the character of information which it has yielded in clinical conditions, the conditions under which the same information may be gained by simpler methods, the conditions under which its employment is essential and those under which it is superfluous.

The failure of the heart has been traced through its varying stages from the simple fatigue of the normal heart in exercise, through the stage of primary overstrain, to that of broken compensation, especial attention being devoted to the states of broken pulmonary compensation arising from failure of the left ventricle and of broken systemic compensation from failure of the right.

The pathogenesis of cardiac symptoms is fully discussed, with their pathological physiology, occurrence, and the symptomatic treatment for their relief.

The general methods of treatment in cardiac diseases, dietetic, pharmacological, gymnastic, hydrotherapeutic, and electrical, have been treated both as empirical procedures and as experimental methods to correct definite disturbances in the physiology of the circulation, especially changes in cardiac force, cardiac tonicity, and peripheral resistance.

The chapters upon the individual organic lesions include discussions of pathological anatomy, pathogenesis, pathological physiology, as well as of symptomatology, course, notes of typical cases, diagnosis, treatment, and prognosis. Considerable attention is also paid to functional disturbances (valvular insufficiencies, etc.) which may bring about conditions similar to those resulting from organic changes or may accompany the latter. The Adams-Stokes syndrome seems so definitely associated with lesions of the auriculoventricular muscle bundles as to justify its classification among conditions due to organic lesions.

The congenital heart lesions are viewed as disturbances in embryologic development in which primary malformations or states in fetal life have diverted the blood current, modifying the further course of development and producing concomitant secondary malformations. The effect of these lesions upon the adult circulation and their relation to cardiac overstrain in producing the syndrome of the morbus cæruleus are discussed, as well as the signs, diagnosis, prognosis, and treatment.

Short chapters are devoted to the subjects of pregnancy in heart disease and the effects of trauma and wounds of the heart.

Considerable space is given to the purely functional disturbances of cardiac action, especially to the physiological mechanisms by which many of them result from disturbances in distant organs as well as to the improvements resulting when these disturbances are corrected.

A great deal of care has been bestowed by the writer in the preparation of the illustrations, especially upon the cardiosphygmographic tracings, the diagrammatic representations of clinical conditions and of effects upon the blood flow in different parts of the circulation as well as in different stages of the disease. When necessary, figures have been borrowed from other sources, to whom due credit has been given.

Since the aim of the book is not only to present the principal facts but to aid the reader in following out lines in which he is especially interested, an adequate bibliography has been added to each chapter, embracing the articles referred to in the text.

It is a pleasant duty for the writer, in conclusion, to express his thanks to Professors Barker and Thayer for the privilege of using the clinical

material and records of the Johns Hopkins Hospital, to Professor T. B. Futcher for that of the Johns Hopkins Dispensary, and to his father, Professor J. O. Hirschfelder, for the cases at the City and County Hospital of San Francisco; to Professor F. P. Mall and Drs. Knower, Retzer, and Evans in matters of anatomy and embryology; to Professors W. H. Howell, J. Loeb, and Dr. D. R. Hooker in physiology; to Professors W. S. Halsted, T. S. Cullen, and J. M. Slemons in matters of surgery, gynæcology, and obstetrics; to Professors W. G. MacCallum and W. Ophüls, as well as to Major F. F. Russell, Dr. Lamb, and Dr. Gray, of the Army Medical Museum, for the use of pathological material; to Professor C. M. Cooper for the collection of radiographs; to Dr. Chas. S. Bond for his untiring labors in the preparation of photomicrographs; to Professor W. Einthoven of Leyden for the use of electrocardiograms; to Professor Max Broedel for his kind instruction and suggestions in matters of illustration; to Dr. Caroline B. Towles for her assistance in reading of proof as well as for many helpful suggestions; and to Miss Alberta E. Bush for her care in the technical matters pertaining to the manuscript and index.

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SYNONYMOUS ANATOMICAL TERMS.

Old Terminology.	Basle Anatomical Nomenclature (BNA)[1].	Latin.
Auriculoventricular groove	Coronary sulcus	Sulcus coronarius.
Interventricular septum	Septum of ventricles	Septum ventriculorum.
Muscular septum		Septum musculare.
Membranous septum		Septum membranaceum.
Auricle	Forechamber	Atrium.
Auricular appendix	Auricle	Auricula.
Interauricular septum	Septum of atria	Septum atriorum.
Columnæ carneæ	Fleshy cords	Trabeculae carneae.
Annulus ovalis	Edge of oval fossa	Limbus fossae ovalis (Vieussenii).
Intervenous tubercle of Lower		Tuberculum intervenosum (Loweri).
Eustachian valve	Valve of inferior vena cava	Valvula venae cavae (inferioris, Eustachii).
Valve of Thebesius; coronary valve	Value of coronary sinus	Valvula sinus coronarii (Thebesii).
Foramina Thebesii	Foramina of the smallest veins	Foramina venarum minimarum (Thebesii).
Tricuspid valve (right auriculoventricular valve)	Tricuspid valve	Valvula tricuspidalis.
Infundibular cusp	Anterior cusp	Cuspis anterior.
Marginal cusp	Posterior cusp	Cuspis posterior.
Septal cusp	Medial cusp	Cuspis medialis.
Left auriculoventricular valve	Bicuspid or mitral valve	Valvula bicuspidalis (mitralis).
Corpora Arantii	Nodules of the semilunar valves	Noduli valvularum semilunarium.
Ductus arteriosus (Botalli)	Arterial duct	Ductus arteriosus (Botalli).
Auriculoventricular bundle (Kent, His)	Atrioventricular bundle (His)	Fasciculus atrioventricularis.

[1] Quoted from Barker, L. F., Anatomical Terminology, with special reference to the BNA, Phila., 1907.

The Basle Anatomical Nomenclature (BNA) is the terminology adopted by an international convention of anatomists at Basle in 1895, for the purpose of securing uniformity of terminology. As it has not yet supplanted the old terminology in clinical usage, the latter is adhered to in this book, though the BNA terms are frequently given in parentheses.

FIG. 3.—Relations of the heart and great vessels, viewed from the front. *S V C*, superior vena cava; *R A*, right auricle (atrium); *L V*, left ventricle; *R V*, right ventricle; *P A*, pulmonary artery; *PL*, pleura.

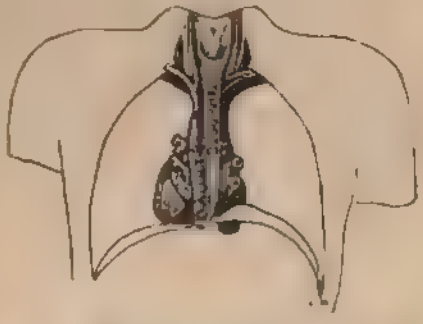

FIG. 4.—The heart and thoracic viscera viewed from behind. The lungs have been cut away. *L A*, left auricle; *L V*, left ventricle.

FIG. 5.—Sagittal section of the thorax viewed from the right. *AZ* great azygos vein; *POST. MEDIAST.* posterior mediastinum; *ANTER. MEDIAST.* anterior mediastinum; *R A* right auricle; *PHREN.* right phrenic nerve.

DISEASES
OF THE
HEART AND AORTA

PART I.
GENERAL CONSIDERATIONS AND METHODS OF DIAGNOSIS.

I.
PHYSIOLOGICAL CONSIDERATIONS.

PROPERTIES OF HEART MUSCLE.

The heart is composed of striated muscle-fibres which differ anatomically from the skeletal muscles in being almost devoid of connective-tissue sheaths and from most of the skeletal muscles[1] in the fact that they anastomose freely with one another, forming a continuous meshwork of muscle

Fig. 6.—Heart muscle-fibres, ×375. (After Pierson.)

tissue (Figs. 6 and 7). Physiologically heart muscle differs from skeletal muscle, for (1) it is continually undergoing rhythmic contractions, and (2), as Bowditch has shown, every contraction is maximal.

A great deal of perspicuity has been added, especially to the clinical study of the cardiac function, by discriminating between influences which affect the cardinal properties of the cardiac muscle (Engelmann). One recognizes those which affect (1) rhythmicity (chronotropic in-

[1] The tongue of the frog and some other forms of muscle somewhat resemble heart muscle in structure.

fluences); (2) irritability (bathmotropic), (3) conductivity (dromotropic); (4) contractility (inotropic), as well as (5) tonicity (Mackenzie).

Influences improving these properties are designated as positive, those which depress them as negative.

FIG. 7.—Section through the endocardium showing cross-section of the muscle-fibres. (After Piersol.)

ORIGIN OF THE HEART-BEAT.

Rôle of the Salts.—Merunowicz, under Ludwig's direction, demonstrated that the rhythmicity of the heart depended not only upon its intrinsic characteristics but particularly upon the action of the inorganic salts present in the blood serum. Ringer (1882), and later Howell, showed that the antagonistic actions of potassium and calcium salts were the factors chiefly concerned in determining the rhythm of the heart, while Loeb and his pupil, Lingle, showed that without the sodium salts it would not beat at all. Accordingly, as Loeb and Howell agree, the heart-beat can be maintained only when these three salts or their ions are present in certain definite proportions, or in what Loeb has termed "a balanced solution." However, while Ringer and Howell believe that the calcium liberates the motive power of the cardiac contraction, Loeb and his pupils believe that this is done by the sodium and that the calcium and potassium merely keep the sodium from liberating too much.

Their mode of action has been explained by Loeb in 1899 in the following words:

"The salts or electrolytes in general do not exist in living tissues as such exclusively, but are partly in combination with proteids (or fatty acids). The salts or electrolytes do not enter into this combination as a whole, but through their ions. The great importance of these ion-proteid combinations (or soaps) lies in the fact that by substitution of one ion for another, the physical properties of the proteid change (*e.g.*, their surface tension their power to absorb water or their viscosity or state of matter). We thus possess in these ion-proteid or soap compounds essential constituents of living matter, which can be modified at desire, and hence enable us to vary and control the life phenomena themselves.

"Life phenomena, and especially irritability depend upon the presence in the tissues of a number of the various metal proteids, or soaps (Na, Ca, K and Mg) in definite proportions . . . Solutions of Na-salts produce rhythmical contractions only if the muscle cells contain

Ca-ions in sufficient numbers. As soon as there is a lack of Ca-ions in the tissues the Na-ions are no longer able to cause rhythmical contractions. On the other hand, if we add Ca-salts in sufficient quantity to the NaCl solution, it will no longer cause rhythmical contractions in the fresh muscle of the frog. . . . It is hardly necessary to mention that this suggested the possibility that molecular contraction in general is due to a substitution of Na for Ca, or *vice versa*, in certain compounds (proteins or soaps, in the muscle."

The hypothesis that the main physiological antagonism lies between K and Ca is stated by Howell in the following words:

"The well-nourished heart contains a large supply of energy-yielding material which is in a stable form, so that it neither dissociates spontaneously nor can be made to do so by the action of external stimuli. It is possible that this stable non-dissociable form consists of a combination between it and the potassium or the potassium salts, and that therein lies the functional importance of the potassium contained in the tissue. This compound reacts with the calcium or with the calcium and sodium salts and a portion of the potassium is replaced, and a compound is formed which is unstable. At the end of the diastolic period this compound reaches a condition of instability such that it dissociates spontaneously, giving rise to the chain of events that culminates in the normal systole. Before spontaneous dissociation occurs it may be hastened by an external stimulus, as we know is the case when a mechanical or electrical shock is applied to the heart at any time after diastole begins."

Any single ion or salt is poisonous by itself, but in the presence of certain others may be beneficial. This very interesting question of "balanced ion solutions" has been extensively investigated by Loeb and his pupils, not only upon heart but upon skeletal muscle and upon lower medusæ, molluscs, and fishes.

Even the mammalian heart can be readily revived and kept beating outside the body if perfused with a solution containing these substances together with sodium bicarbonate (Howell) and saturated with oxygen (Locke's solution,—NaCl 0.9 per cent. + $CaCl_2$ 0.024 per cent. + KCl 0.42 + $NaHCO_3$ 0.01 to 0.03 + dextrose 0.1 per cent.). It is necessary to maintain the blood-pressure at 50–100 mm. Hg, and also the temperature 36° to 37°. Kuliabko and others have revived excised human hearts many hours after death. Fibrillary contractions occasionally set in, but may be stopped by perfusing with KCl 1.0 per cent. for a few minutes instead of Locke's solution. The heart then comes to a stand-still and resumes beating under Locke's solution.

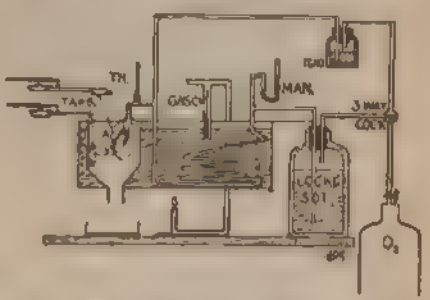

FIG. 8.—Apparatus for perfusing the mammalian heart. *A*, auricle, *V*, ventricle, *TAMB*, tambour, *TH* thermometer, *MAN.*, manometer; *GASCH*, gas check, O_2, tank of oxygen.

The study of the excised heart has been very useful both in testing the effect of drugs and in simulating conditions of disease; but the conditions of circulation are not exactly comparable to those within the animal, and the results should always be carefully checked upon the intact animal before assuming them to be normal or drawing any conclusions as to pharmacological action.

Myogenic and Neurogenic Theories.—Whether the salts or ions which maintain the rhythmicity of the heart-beat do so by acting directly upon

the muscle tissue (myogenic), or whether the stimuli are first generated in nerve tissue (neurogenic) and then transmitted to the muscle, is a question which has been disputed for centuries. And though the pendulum has repeatedly swung from one opinion to the other, this question cannot at present be answered. It is quite certain that all the extrinsic cardiac nerves can be removed without stopping the rhythmic contractions, and that the ganglion cells may be stimulated without materially affecting the rhythm (Gaskell). But the meshwork of muscle-fibres in the heart is so permeated by a meshwork of fine nerve-fibres that it has been impossible to determine whether the impulse arises in the muscle-cells or in the nerve endings upon their surfaces. Wm. His, Jr., has indeed shown that the heart of the chick embryo beats before nerve-fibres have entered it at all, but the possibility still remains that after once entering the heart the nerves may take the initiation of contraction away from the adult heart-muscle. Moreover, the recent experiments of Carlson and of Magnus in allied fields give considerable evidence that such may be the case; so that, in spite of its importance for both the physiology and the pathology of the heart, neither the myogenic nor the neurogenic theory of the heart-beat has been finally proved.

Maximal Contractions and Irritability.—As Bowditch has shown, the heart liberates all its available energy at each contraction, which resembles in this way the explosion of gunpowder or the liberation of a spring by a trigger. Like the power of the spring, the strength of the cardiac contraction depends upon the energy stored up. This energy seems to depend upon the regeneration of the contractile substance mentioned above by Howell. When the next contraction, normal or abnormal (extrasystole), occurs soon after the last (early in diastole), the contraction is weaker than the preceding, since it liberates less energy, but the contractile substance is again completely destroyed and requires another pause (compensatory pause, see page 69) to regenerate it. When it occurs late, the contraction is of almost or quite original strength, and the stored-up energy is again liberated completely. Moreover, Erlanger has shown that the irritability of the heart increases progressively as diastole is prolonged and as the muscle becomes overloaded with the energy-producing substance.

ORIGIN AND COURSE OF THE CARDIAC IMPULSE.

The Sinus as "Pace-maker" of the Heart.—In the frog, where the cardiac impulse travels slowly, it is very easy to see that it arises at the sinus venosus, which executes a contraction. This is followed by contraction of the auricle, the latter after an appreciable interval by a visible contraction of the small ring of muscle about the auriculoventricular ring (Bond), and this in turn by contraction of the ventricles.

It is probable that the sinus initiates the cardiac rhythm, because it is the chamber which, when isolated, beats at the fastest rhythm in the blood-serum, and hence it becomes what Erlanger terms "the pace-maker of the heart."[1] Indeed, if the impulse from the sinus is blocked by crushing

[1] Under pathological conditions and especially in the excised heart the ventricle may become more irritable and may become the pace-maker (reversed rhythm).

or by cooling the sino-auricular border, the impulses no longer reach the auricles, which must then contract by their own slower rhythm or not beat at all (sinu-auricular heart-block).

Anatomy of the Sinus Region in Mammals.—In man and other mammals the sinus no longer exists as a separate chamber, though in the early embryo (Fig. 243, page 425) its homologue, the sinus reuniens, is separated off from the rest of the auricular cavity by the Eustachian valve. This sinus chamber receives the two venæ cavæ and the coronary sinus. In the course of development the growth of the sinus region does not keep pace with that of the auricle, and it becomes swallowed up in the latter, so that in the adult the sinus corresponds roughly to the area bounded by the mouths of the two venae cavae, the coronary sinus, and the interauricular septum.

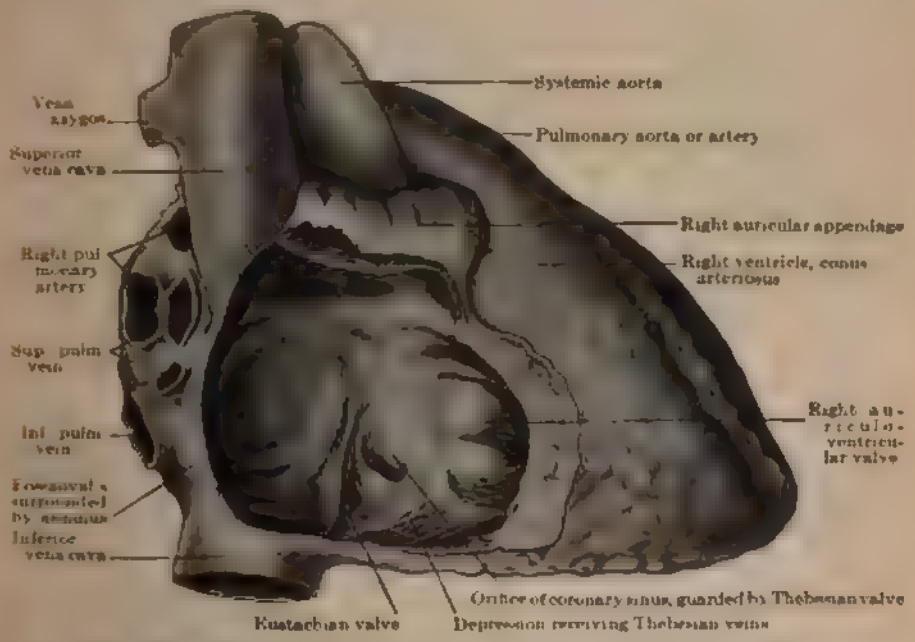

FIG. 9.—The auricular end of the human heart viewed from the right. (After Piersol.)

Comparatively little is known concerning the structure of this important region. The most careful studies (Keith, Retzer, Schonberg) are concerned more with the structure of the veno-auricular junction or the sino-ventricular connections than with the structure of the sinus as a whole, or the sinu-auricular border which is included within the body of the right auricle.

The Veno-auricular Junction.—Schönberg studied the veno-auricular junction in a large number of normal and abnormal human hearts by means of serial sections, each series being composed of 300 to 800 sections. At a level 10 to 15 mm. above the entrance of the superior vena cava into the auricle (atrium) he found the usual structure of vein wall. Below this level the media is found to contain groups of striated muscle-fibres separated from one another by fat and connective tissue. These striated muscle-fibres arise in the

vicinity of non-striated fibres but are never continuous with them. Bundles of these fibres ½ to 1 mm. in diameter run transversely across the vein toward the auricle, gradually converging into larger bundles, which are separated from one another by a tissue rich in lymph- and blood-vessels. In the angle (sulcus) formed between the auricle (atrium) and vena cava these bands of striated muscle become much thinner and contain numerous tortuous fibres resembling Purkinje fibres. In this region there is a considerable deposit of fat, lymphoid and connective tissue, forming a more or less definite border-line. The muscle-fibres of the auricle (atrium) are inserted in the connective tissue here. The connection between the musculature of the vena cava and that of the auricle is made by the numerous small bundles of striated muscle-fibres lying just beneath the endocardium, which pass across this junction and end in the fibres of auricular muscle. "In the macroscopic preparations it is almost always readily seen that the sulcus is bridged at its posterior lateral third by a muscle-bundle which ascends upwards and backwards from the auricle (atrium) to the superior vena cava, where it is strengthened by fibres from the

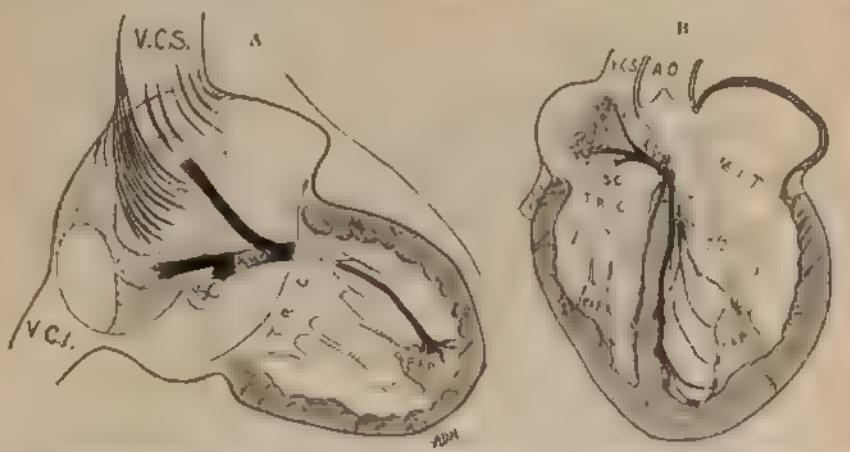

FIG. 10.—The sinus region of the heart, the veno-auricular or veno-sinal bands of striated muscle and the auriculo-atrio-ventricular or sino-ventricular muscle bundle. Schematic, constructed from the findings of Keith, Schönberg and Retzer. A. Seen from the right side. The dotted area represents the sinus region; the strize represent the veno-auricular muscle strands. *V.C.S.*, superior vena cava; *V.C.I.*, inferior vena cava; *SC*, coronary sinus; *A.V.B.*, auriculo-atrio-ventricular muscle bundle (His bundle); *TRIC*, tricuspid valve; *PAP*, papillary muscle. B. The same region seen from the front. *M.I.T.*, mitral valve; *A.O.*, aorta.

circular musculature of the lower part of the vein. This bundle is also well seen macroscopically, but numerous other smaller muscle bundles are seen as well. It corresponds quite well with that described by Keith and Flack and Wenckebach.

Schönberg found that the region of the sulcus is particularly rich in nerve-fibres, ganglion cells, blood-vessels and lymphoid tissue, and is therefore particularly liable to pathological infiltrations and cicatrizations.

It is worthy of note that the sulcus noted by Schönberg does not represent the sinu-auricular junction but the veno-sinal junction. The strands of striated muscle which he describes are derived from the sinus. The sinu-auricular (sinu-atrial) junction on the other hand is actually situated within the body of the auricle (atrium).

Rôle of the Sinus in Mammals. There is a considerable amount of physiological as well as anatomical evidence that in the adult mammal as well as in the amphibian this is the region in which the cardiac impulse arises.

MacWilliam in 1888 was able to show that this intervenous area was the only region at which the application of heat quickened and cold slowed the heart rate. Adam and the writer were able to confirm this observation. H. E. Hering has shown that this area is often the last to cease contraction in dying mammalian and human hearts, though this is not always the case (Hirschfelder and Lyster). Langendorff and Lehmann and also Leon Fredericq showed that even in the excised heart portions of the auricles cut off from this area ceased to beat or beat at a slow rhythm, while those which remained attached to the sinus region beat at about the original rate. Erlanger and Blackmann were able to produce halving of the heart rate (sino-auricular block?) by torsion of this area in the excised heart, but like Hirschfelder and Lyster were unable to produce it by clamping experiments upon the heart *in situ*. The most conclusive experiments are those of Lohmann who poisoned the cells in this area by direct application of cotton soaked in formalin, and found that the heart at once slowed, auricles no longer followed ventricles, and the auricles and ventricles beat simultaneously (nodal rhythm, see page 76).

Course of the Impulse after Leaving the Sinus. — From the sinus region the cardiac impulse travels to the walls of the auricles and gives rise to the auricular contraction. It is also propagated downward toward the ventricles, which it reaches about one-fifth of a second later.

It is a mooted point at present whether the path from sinus to ventricle is through auricular tissue or whether there is a direct sino-ventricular pathway, as believed by Retzer, who thinks that the auricle is off on a side path and contracts first merely because it is nearer to the sinus than is the ventricle. However, Bond's observations on the frog, showing that the auricle contracts a considerable time before the musculature of the auriculoventricular ring, indicates that the impulse passes from the former to the latter.

Kent, His, Retzer, Braeunig, Keith, and Tawara have shown that the cardiac impulse is propagated from auricles to ventricles through the system of Purkinje fibres, which forms a Δ whose shaft arises in the right auricle at or near the sinus, runs in the membranous septum (auriculoventricular bundle) downward to the muscle septum, where it divides into two branches which straddle the muscular septum and then pass to the right and left ventricles. Within these chambers the branches divide into numerous ramifications which lie just beneath the endocardium and pass downward as a meshwork of light-colored translucent strands to the papillary muscles and walls of the ventricles. Occasionally instead of following the walls they cross the ventricular cavity to the papillary muscle as isolated strands (moderator bands, T. W. King, Tawara). In this bundle also the presence of numerous nerve-fibres (Tawara) and of ganglion cells (Gordon Wilson) renders it doubtful whether the impulse travels through nerve or muscle. The slow time of transmission is a little in favor of the latter.

COÖRDINATION OF THE CARDIAC CHAMBERS.

Under all circumstances (except those mentioned on page 67) the contractions of both auricles and of both ventricles are absolutely synchronous. Barker and Hirschfelder have shown that simultaneous contractions of the two ventricles continue after the branch of the conduction system to one (the left ventricle) has been cut, and hence the coördination does not depend upon the auriculoventricular conduction system but upon the ventricular musculature.

Anatomy of the Ventricular Muscle.—This is not surprising, since, as Ludwig, Krehl, and J. B. MacCallum have shown, each strand of muscle-fibres passes from ventricle to ventricle. These muscle-fibres are arranged in three distinct layers so placed that they are wound up like a scroll, the most superficial layer of the left ventricle penetrating to become the deepest layer of the right (Fig. 11, I, II, III). Besides these MacCallum has described a fourth band of muscle, independent of the latter, which surrounds both the aortic and mitral orifices in a single ring of muscle (mitro-aortic ring), across which a septum of connective tissue separates the aortic orifice from the mitral ring (Fig. 11, V). This band is more or less homologous to the bulbus arteriosus of the lower vertebrates, and plays a most important rôle in preventing leaks at the valvular orifices.

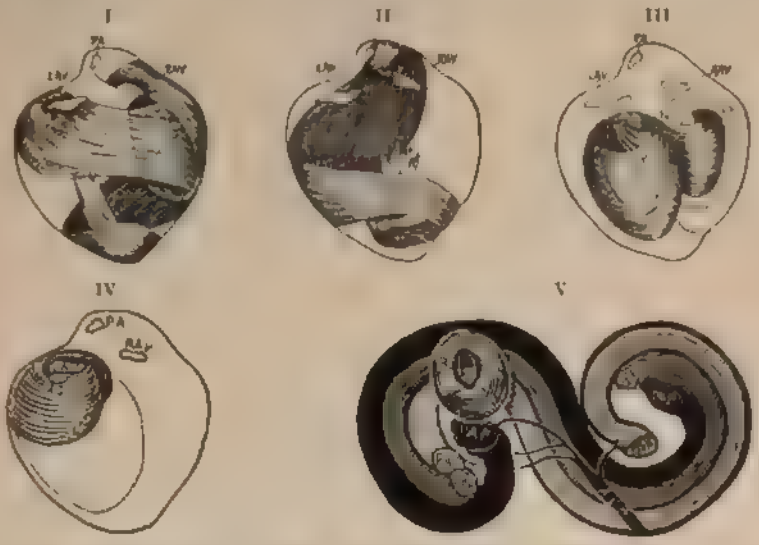

FIG. 11.—Arrangement of ventricular muscle-fibres. (After MacCallum. I and II, superficial fibres of the left ventricle and conus arteriosus. III, deep layers of the left ventricle; IV, rings of muscle about the aortic and mitral orifices, V, diagram representing these relations. *MAV*, mitral orifice, *RAV*, tricuspid orifice, *PA* pulmonary artery, *AO*, aorta *PAP*, papillary muscle. *AVB*, auriculo-(atrio)ventricular bundle, *AORTIC*, ring of muscle-fibres surrounding both the aortic and mitral orifices (mitro-aortic ring).

EMPTYING AND FILLING OF THE HEART, AND MOVEMENTS OF THE VALVES.

The Presphygmic Period.—The instant before the beginning of ventricular systole the mitral and tricuspid valves are open, while the aortic and pulmonic valves are closed. When the ventricular contraction begins, it at once raises the pressure within the ventricles above that in the auricles, causing the mitral and tricuspid valves to close with a snap. There is thus a short interval, the presphygmic (.07–.09 sec.), at the very beginning of systole, during which all four valves are closed and movement of blood ceases in all four chambers. This period lasts until the pressure within the ventricles rises above the arterial pressures (minimal pressure), after which the blood is driven out during the rest of systole.

Method of Recording the Volume Curve.—Yandell Henderson has recorded the emptying and filling of the ventricles by means of a specially constructed cardiac plethysmograph or cardiometer like that of Tigerstedt and Johannson. Henderson's cardiometer was made from an ordinary rubber ball, out of which a large window was cut and then closed hermetically by cementing on a curtain of rubber dam. In the centre of the rubber dam a hole was cut just large enough for it to fit air-tight in the auriculoventricular groove. The heart was then pushed in through the hole until the dam slipped into the groove. The changes of pressure within the air space surrounding the heart were communicated to a recording tambour through a glass tube cemented in the opposite surface of the ball (Fig. 12). Dr Cameron and the writer have found it most convenient to have the recording tambour inverted, so that upstrokes record systole and downstrokes diastole, while a general rise in the curve indicates diminution in volume, and a general fall indicates dilatation.

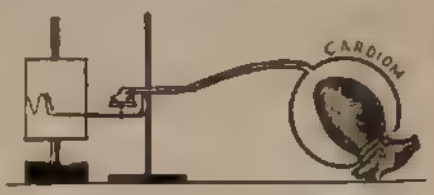

FIG. 12.—Apparatus for registering the volume of the ventricles. *CARDIOM.*, cardiometer.

Outflow during Systole.—By this means Henderson has found that during systole the ventricles do not empty themselves with a rush at the beginning of systole, but that the outflow continues quite uniform throughout at least nine-tenths of the latter period (outlasting the rise of the arterial pulse-wave) and begins to slow only toward the very end (slight rounding of the crest of the curve). At the cessation of outflow there is an instant during which the ventricular pressure is falling, in which no inflow takes place, but this is only one or two hundredths of a second and is difficult to estimate accurately. This instant corresponds to the dicrotic notch upon the aortic pulse-wave.

Filling of the Ventricles.—The ventricles then begin to fill at a rapid and uniform rate until they are almost completely distended. If the pulse-rate is rapid, the next systole takes place before the filling is as complete as possible, and cutting short the filling diminishes the volume of the heart; not only the total volume, but the amount of blood discharged at each systole (Fig. 13).

FIG. 13.—Volume curves of the ventricles at different heart rates. (Modified from Henderson.) The dotted line shows how the curve of the typical cycle may be superposed upon the curve corresponding to a different rate. *A*, quota of blood forced in by auricular systole.

Diastole and Diastasis.—If, on the other hand, the heart rate is slow (Fig. 13), as after stimulation of the vagus, the influx begins at the same rate as before and continues uniformly for about two-fifths of a second (steep ascent of the curve) until the ventricles are distended, after which scarcely any blood flows into the ventricles no matter how long the interval

to the next beat. The diastolic period is thus divided into two parts: (1) the phase of diastole proper during which filling of the ventricles takes place; (2) the phase of diastasis in which little or no filling occurs. The slower the heart the greater is the diastolic filling and the longer its duration. The greatest amount of output in unit time occurs at a rate which just allows the phase of diastolic filling to be complete but in which the next beat occurs before diastasis sets in. Any rate above or below this brings about some slowing of the circulation.

Position of the Valves in Diastole. Baumgarten (1843) has been able to demonstrate upon the excised heart that the cusps of the mitral and tricuspid valves are floated together by the influx of blood and the valves close spontaneously when the inflow ceases. The writer has been able to show that the occurrence of diastasis is not necessarily caused by the valves being closed, but by the fact that the heart fills for a time before the walls are

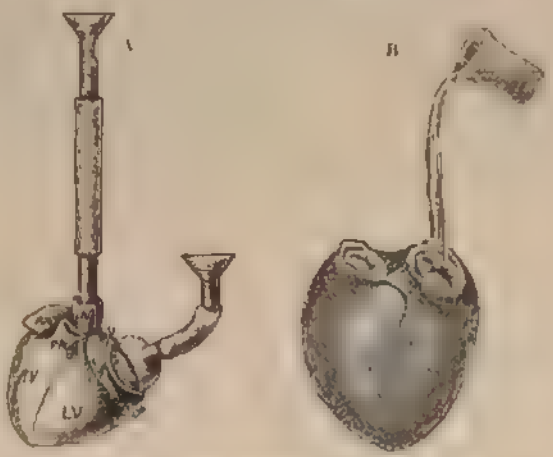

Fig. 14.—Methods for demonstrating the movements of the heart valves. A, Gad's method; B, Baumgarten's method.

put upon a stretch, and then the passive elasticity of the walls prevents further filling. If the venous pressure is materially increased, further increase in volume then takes place. The closure of the valves in early diastole depends chiefly upon the suddenness both of the filling and of its cessation.

A very pretty and instructive demonstration of the opening and closing of the heart valves has been devised by J. Gad by an experiment shown in Fig. 14, A, which can be very nicely performed upon a sheep's heart as bought at a butcher's shop.

The left auricle is cut away and the bowl of a large thistle tube tied in the place by a circular ligature. A large glass tube is thrust through the aorta into the ventricle and ligatured in place. Both the thistle tube and the aortic cannula are connected with funnels by means of rubber tubes, and the chambers of the heart may be then completely filled with water. The opening and closing of the valves may be brought about by raising and lowering one or the other of the funnels, and may be watched through the wall of the bulb. Insufficiency of the valve may be produced by cutting or stretching one of the chordæ tendineæ, but after the experiment has been repeated a few times upon the same heart a certain amount of insufficiency usually sets in spontaneously. The sounds produced by the valves and blood stream independently of the contraction can be well studied by placing the stethoscope upon such a heart, provided all the air has been removed from the cardiac chambers. In a similar manner the phenomena can be observed in the right

heart. The closure of the aortic or pulmonic valves can also be demonstrated by drawing the glass tube out of the ventricle up into the vessel tying it there close to the valves, and cutting off the walls of the vessel above the ligature.

Still simpler is the older method of Baumgarten (1843) of cutting away the auricles to expose the valves and then pouring in water from a beaker (Fig. 14, B).

RELAXATION OF THE HEART, AND TONICITY OF THE CARDIAC MUSCLE.

It has been supposed by some writers that the diastolic dilatation of the heart is brought about by some active muscular contraction, since the pressure within both ventricles becomes negative, even to the extent of − 55 mm. Hg. This negative pressure is of only momentary duration, and may be compared to that occurring within a rubber ball when squeezed and let go. The walls of the heart are sufficiently rigid and are sufficiently provided with elastic fibres to resume their shape like a rubber ball, and, on the other hand, the pressure in the coronary arteries tends to hold them distended as though by a wire frame.[1]

The heart muscle is quiescent and the heart walls are relaxed during the entire period of diastole, so that neither the most delicate recording levers nor the most sensitive galvanometers reveal the slightest signs of contraction. Nevertheless, as will be seen, the degree of this diastolic relaxation of the walls varies considerably under different circumstances dependent upon the tonicity of the heart muscle. This is shown by variations in the length of strips of cardiac muscle under a constant load, as well as by variations in the cardiac volume.

Tonicity.—Tonicity may be defined as the resistance of the heart muscle to stretching in diastole; or, less accurately, as its diastolic rigidity.

The force which stretches the heart walls in diastole is the pressure at which the blood enters the heart from the great veins, namely the venous pressure, so that with a high venous pressure (unless antagonized by a high tonicity) they will be stretched considerably (dilatation), while with a low venous pressure comparatively little blood will enter and the heart will remain small. In all cases filling will continue until an equilibrium is reached between the venous pressure and the cardiac tonicity, unless the heart rate is so rapid that the filling is interrupted by the next systole. A high tonicity will, however, antagonize a high venous pressure and prevent overfilling.

Moreover, Howell and Donaldson have shown that the systolic output of the heart depends to a great extent upon the amount entering the latter from the great veins, and hence, upon the venous pressure. If the venous pressure falls below a certain level, the heart fills incompletely, and the ventricles are unable to pump enough blood into the arteries to maintain the blood-pressure at the usual level.

The rate of filling of the heart is accelerated (curve of filling steeper) (Fig. 13) when either the venous pressure is high or the tonicity is low; the filling is slowed (curve more oblique) when either the tonicity is

[1] For a detailed account of the various theories of the cardiac relaxation, with full bibliography, consult E. Ebstein, Die Diastole des Herzens, Ergebnisse der Physiol., Wiesb., 1904, iii, 2 Abth.

high or the venous pressure is low. So that, as regards filling of the heart, a high tonicity is equivalent to a low venous pressure, and conversely, a low tonicity is equivalent to a high venous pressure (Fig. 16).

Influences which affect tonicity may be studied objectively in isolated strips of cardiac muscle by means of their shortening or lengthening, or upon the intact heart by changes in the volume curve.

Fig. 15.—Volume curves showing the effect of variations in venous pressure (*V. P.*) and in tonicity (*T*) upon the rate at which the ventricles are filled during diastole.

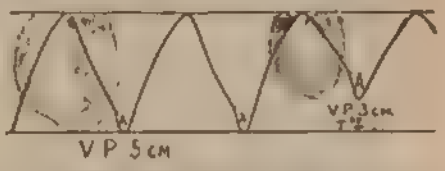

Fig. 16. Volume curves showing the effect of low venous pressure or of high tonicity upon the amount of blood entering the ventricles. (Modified from Y. Henderson.) A fall in the venous pressure is equivalent to an increased tonicity. *CM*, centimetres of H_2O pressure.

The total volume of the heart at any given instant may be regarded as follows:

Volume of heart = volume of heart walls + volume of blood within cardiac chambers.
Volume of walls = volume of muscle + coronary blood + lymph. (The two latter factors vary somewhat, though relatively slightly, the lymph increasing considerably in cardiac stasis.)
Volume of blood within chambers = output at each systole + blood remaining at end of systole (residual blood).

Residual Blood. The residual blood undergoes great variations. In dilated hearts it may attain to several times the amount of the systolic output (cf. Fig. 17), while in small hearts it may be only a fraction of the latter. The systolic output, on the other hand, may undergo equally large variations.

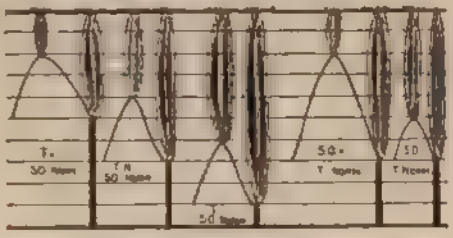

Fig. 17. Diagram to illustrate the changes in volume of the ventricles in systole and diastole associated with variations in tonicity. *T* and systolic output. *S. O.* *N. NORM.*, normal. +, increased. —, diminished. Length of heavy black line indicates degree of tonicity.

The changes in tonicity may be measured by the volume of the heart at the end of diastole, i.e. when the filling is most complete, a large diastolic volume representing low tonicity (when venous pressure and pulse-rate are constant), a small volume indicating a high tonicity.

Nature of Changes in Tonicity.—Porter has found that a strip of heart muscle can be made to remain elongated (diminished tone), or can then be made to remain shortened when not receiving any stimuli whatever (increased tone). Several degrees of this permanent shortening can be super-

posed on one another with great similarity to the tetanus of skeletal muscle ("tetanus of tone," Porter). Barcroft and Dixon have shown that the muscle when in tone gives off more CO_2 than when at rest, further supporting this view of the rôle of increase and decrease in tone.

Factors producing Changes in Tonus.—F. B. Hoffmann has demonstrated that there are two separate sets of fibres in the frog's vagus. One set influences the heart rate only (chronotropic effect), the other increases the size and force of contraction (augmentor effect) and also increases the cardiac tonus but does not affect the rate at all. This group of fibres is found only in the interauricular and interventricular septum (septal nerves) in the frog. In other animals the two groups of fibres pass side by side and cannot be dissociated, though it is frequent in weak stimulation of the vagus to find one effect occurring without the other.

P. D. Cameron, in the writer's laboratory, has found that in dogs the intravenous administration of digitalis, strophanthus, nitroglycerin, and calcium salts increases cardiac tonicity. The effect of small (therapeutic) doses of these drugs is exerted almost entirely upon the tonic fibres in the vagus, and fails to appear if the vagi have been cut or paralyzed with atropine. Larger doses, however, exert similar effects by direct action on the heart muscle. Atropine itself illustrates these effects by causing a primary depression of tonus as the vagi become paralyzed, which is followed by an increase in tonicity from direct action on the heart muscle. Potassium salts, asphyxia, formic acid, adrenalin depress tonicity. Aconite in therapeutic doses affects rate more than tonus in the dog.

Since the exact volume of the heart cannot be determined clinically, the area of the cardiac shadow in diastole furnishes the best index of the tonus, especially when combined with study of the venous pressure. Comparatively little investigation has been carried on in this field. Moritz and Dietlen have shown that exercise usually increases tonus in healthy persons. The study of tonus has also proved of value in the study of exercise and in the controlling of hydrotherapy and drug treatments, as well as in the study of myocardial insufficiency.

ACTION OF THE CARDIAC NERVES

The heart rate is determined by action of the vagus and accelerator nerves, and particularly by the tonic activity of the centres near the calamus scriptorius of the medulla: the former nerve slows the heart (inhibitory effect), lowers the blood-pressure (depressor effect), and *diminishes the conductivity* (negatively dromotropic effect) from auricle (atrium) to ventricle; the latter quickens the heart (*accelerator effect*), increases the force of the contraction and cardiac tonus (*augmentor*), and improves conductivity (Bayliss and Starling). In some cases stimulation of the accelerators may revive a heart that has ceased to beat (Hering).

Both vagi and accelerators are normally in tonic activity. Reflex quickening of the pulse-rate, as from emotion, pain, sensation, and other reflex causes (Reid Hunt), and moderate exercise (Hering and Bowen), is due partly to diminution of tonic activity of the

vagi, partly to direct stimulation of the accelerators (Hooker); while the acceleration after violent exercise is due to stimulation of the accelerators (Hering, Bowen). Acceleration upon mild exercise can also be obtained in patients whose vagi are made inactive by 0.5 to 1.0 mg. ($\frac{1}{120}$ to $\frac{1}{60}$ gr.) atropine (Hirschfelder). On the other hand, exercise caused no acceleration but a slight slowing of the pulse in a dog from which Friedenthal had removed all the cardiac nerves.

FIG. 18.—Origin and course of the cardiac nerves, and cutaneous distribution of the corresponding branches. Schematic, modified from Douglas Powell and Gibson.) *MOT SENS* nuclei of the efferent (motor, and afferent (sensory) fibres of the vagus. *C* 1, 2, 3, 4, 5, 6, 7, 8, and *T* 1, 2, 3, 4, 5, 6, 7, 8, cervical and thoracic (dorsal) spinal nerves and their cutaneous distribution. *SCG, MCG, ICG* superior, middle, and inferior cervical ganglia. *REC LAR*, recurrent laryngeal nerve; *CPL*, cardiac plexus.

The mode of action of the cardiac nerves has been shrouded in mystery, especially that of the inhibition by the vagus. Howell and Duke in a most brilliant series of researches have shown that potassium is given off from the heart muscle and can be found in increased quantity in the perfusion liquid after the vagus has been stimulated. Controls without vagus stimulation show no such increase. It would therefore appear that vagus inhibition is a true potassium effect, a fact further borne out by the close analogy between the action of this element and stimulation of the vagus, as well as by the marked increase in the action of the vagus after the administration of large quantities of potassium or after increase of potassium in the blood.

These observers were unable to demonstrate any effect of the accelerators upon the liberation of calcium, potassium, or nitrogen.

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II.

BLOOD-PRESSURE AND BLOOD VISCOSITY.

THE BLOOD-PRESSURE IN HEALTH AND DISEASE

Definition.—The blood-pressure, or "arterial tension," is the pressure which the blood is exerting upon the walls of the vessel in which it is to be measured (lateral pressure), or upon the column of blood ahead of it in the direction in which it is flowing (end pressure).

The end pressure is equal to lateral pressure + velocity head, but as a rule differs by only a few millimetres from the lateral pressure. The end pressure in the branch of a vessel is equal to the lateral pressure in the vessel from which it branches.

FIG. 19.—Curve of intraventricular and aortic pressures. (After Huerthle.) *A*, aortic pressure; *V*, intraventricular pressure. *D*, curve, taken with a differential manometer showing the differences of pressure between left ventricle and aorta.

Pressure within the Left Ventricle.—It is evident that in a system of elastic tubes like the arteries, the pressure of the blood in any segment of artery is brought about by the tendency of the inflow (from the heart) to remain in excess of the outflow through the capillaries. The inflow to the arteries is maintained by the pumping action of the heart, that is, by the intraventricular pressure during systole. As shown by Huerthle and Porter the pressure within the ventricle remains tolerably constant throughout systole and takes the form of a plateau more or less independent of the pulse curve in the aorta (Fig. 19), though the floor of the plateau slopes downward with peripheral dilatation and upward with constriction.

If the systole is too weak to open the aortic valves, and the heart contracts without change in volume (isometrically), the curve does not remain a plateau, but has a rounded apex like that of the isometric contraction of skeletal muscle. The pressure within the ventricle when the

aortic valves are open is slightly in excess of that within the aorta, that is very little above the maximal pressure as measured in the latter, and it remains at this height until the end of systole, when the aortic valves close.

Huerthle and others have shown that the intraventricular pressure is not always constant throughout systole, but that when the peripheral resistance is very low it falls toward the end of systole, while if the peripheral resistance is very high it rises toward the end of systole.

The Maximal, Minimal, and Pulse Pressures.—Accordingly, when the aortic valves open (.07 to .09 second) after the beginning of ventricular systole (see Fig. 45, page 53), the pressure in the aorta soon rises to its maximum, and from that time until the beginning of the next systole it diminishes more or less gradually as the excess of blood flows out from the arterial tree through the capillaries and into the veins. The minimal pressure is reached just before the beginning of the next systole. The pulse-pressure is the difference between the maximal and minimal pressures.

Characteristics of the Pulse. It is evident that the maintenance of the circulation depends upon the head of pressure in the arteries, and accordingly much attention was paid by the older clinicians to the "arterial tension" and the "quality of the pulse," which they thought were manifestations of it. The arterial tension was judged by the force necessary to obliterate the pulse at the wrist when the fingers are pressed upon the radial artery.

A still more accurate method of feeling the pulse is to empty the artery for a few centimetres by 'milking' out the blood with two fingers of one hand while obliterating the artery above the wrist with the fingers of the other hand. The pressure of the latter is then gradually diminished until the return of the pulse is felt, this point marking the maximal or systolic pressure. By careful training of the sensations and comparing the observation with the results of a good sphygmomanometric determination made at the same time, a great degree of skill in judging pressures may be attained, one of the writer's teachers, who has cultivated this perception to a remarkable degree, voices the general experience in saying, "I can estimate the blood-pressure with the fingers alone quite accurately in about eight cases out of ten, but those in which it is of real importance are always the other two."

The minimal pressure may also be judged but even less accurately, by estimating the amount of pressure at which the size of the pulse just begins to decrease as one raises the pressure in the artery.

Determination of Maximal Blood-pressure.—Instruments for determining the blood-pressure date from 1855, when K. Vierordt determined the weight that could be placed over the radial artery before the pulse was obliterated.

Marey (1876) devised the first useful apparatus for estimating the blood-pressure in man. He placed the hand in a plethysmograph connected with a bottle for raising the pressure and a sphygmoscope tambour for recording the size of the pulse-waves. He states (1878) that the maximal pressure may be determined as the point where the pulsation disappears, the minimal as the point where the oscillations are largest. It is worthy of note that Marey was twenty-five years in advance of the times, and that his methods and conclusions are almost exactly those of the best modern methods (Erlanger and v. Recklinghausen). Unfortunately the work of Marey was little known, and the first apparatus to attain general use was that of v. Basch (1887). Unlike Marey, v. Basch studied only the maximal pressures, but much good pioneer work was done with this

instrument. It consisted of a small rubber bulb filled with water and communicating with a mercury manometer. The bulb was pressed upon the radial artery until the pulse below it was obliterated and the pressure necessary was read off upon the manometer. V. Basch modified the apparatus later by using a spring manometer, and Potain substituted air for water in the bag with an aneroid barometer. This method is still almost universal in France, but the possible error with v. Basch's as well as Potain's methods is as much as 78 mm. Hg (Tigerstedt).

Riva-Rocci (1896) and L. Hill and H. Barnard (1897) introduced the use of a rubber bag about the upper arm, surrounded by a non-elastic cuff of silk (Riva-Rocci) or of leather (Hill and Barnard) instead of the small bag that Potain pressed upon the artery, and they compressed the artery with pressure from an air-pump or Davidson syringe, feeling the return of the pulse at the wrist as the air was allowed to escape and reading off the pressure corresponding. These are the methods now in most general use, the only modification being that the rubber bag must not be less than 12 cm. in diameter instead of 5 cm. as used by Riva-Rocci; for v. Recklinghausen has shown that with narrow cuffs a great deal of pressure is lost in squeezing the tissues, and hence the readings obtained with them are too high, but this is now remedied by using the broad cuff. Riva-Rocci's method was used only for determining the maximal pressure.

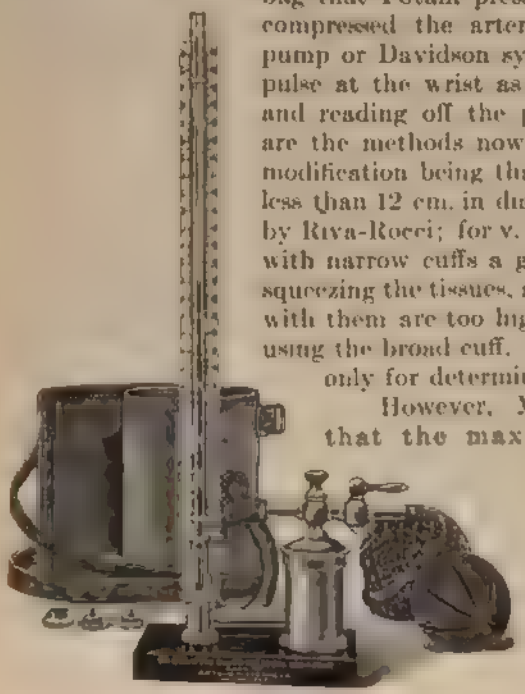

Fig. 20.—Riva-Rocci blood pressure apparatus as modified by Stanton. (By kindness of the A. H. Thomas Co.)

However, Marey (l.c.) had shown that the maximal pulse-wave was obtained when the pressure about an organ was equal to the pressure within the artery supplying it (*i.e.*, the minimal pressure.—Howell and Brush), and this observation furnished a basis for such determinations in man.

Determination of Minimal Blood-pressure.—Numerous methods for determining the minimal blood-pressure have been devised, especially those of Hill and Barnard, and Mosso, but those which are useful and reliable in practice date from 1901, when Masing began to determine minimal pressure by the point at which the radial pulse seemed to become largest. About the same time Janeway estimated the minimal pressure at the point where the oscillations of the mercury column in the manometer seemed greatest. This is a satisfactory method in most cases, but the judgment by the eye is sometimes difficult and in small pulses may be impossible.

In 1903 Strasburger revived Masing's method as did also Sahli, who recorded the maximal pulse-wave with a sphygmograph at the wrist. The latter method is very cumbersome. For practical purposes the method of Masing and Strasburger

is fairly satisfactory, provided a few precautions are taken. First, it is necessary to exert an absolutely uniform pressure with the fingers upon the radial artery throughout the determination. Ordinarily this is very difficult; but if the artery is palpated with the ball of the finger instead of the finger-tips, while the finger-tips rest against the radius, as shown in the figure (Fig. 21), any changes of pressure by the fingers are exerted against the bone and not against the artery, and a very uniform pressure is exerted upon the latter. Secondly, it is necessary to raise and then gradually let out the pressure from the bag while feeling the pulse in this way, in order to acquaint oneself with the changes of pulse to be expected. Thirdly, it is necessary to repeat the determination four to six times in order to eliminate the great discrepancies that creep in when single readings are made. All the non-concordant readings should be disregarded. In this way fairly accurate determinations of minimal pressure may be obtained (within 5 mm. of those obtained by Erlanger's apparatus).

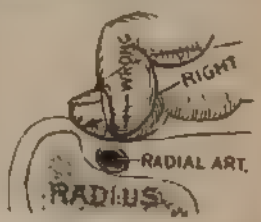

Fig. 21.—Correct method of feeling the pulse in Strasburger's determination of minimal pressure.

Erlanger's Sphygmomanometer.—The most accurate and satisfactory, if somewhat bulky, sphygmomanometer is that of Erlanger with which graphic records of both maximal and minimal pressures may be obtained (Fig. 22). Erlanger's apparatus differs from the Riva-Rocci with Recklinghausen cuff only in the fact that by means of a *T*-tube the cuff is connected also with a rubber pressure-bag in a glass case. The oscillations of pressure in the cuff are thus communicated to the pressure bag, and the oscillations of this bag are communicated to the air in the glass case around it, and are recorded by the movements of a Marey tambour upon the smoked paper on a small drum. He is also able to let the pressure flow out very slowly by a series of capillary outlets of different bores. A complicated stop-cock allows any of these to be used at will.

Fig. 22.—Erlanger blood-pressure apparatus with Hirschfelder polygraph attachment. Kindness of Schneider Bros.

In using the Erlanger apparatus, one turns the stop-cock to the point marked "In," then raises the pressure in the bag to well above the maximum arterial pressure, and turns the stop-cock to the point marked 1 or 2, which corresponds to capillary outlets of different sizes. The pressure in the bag falls gradually, and soon small oscillations of the lever are seen, due to the impact of the compressed artery upon the upper margin of the bag. A sudden increase in the size of these wavelets soon takes place and marks the maximal pressure, which is read off on the manometer; it is just a trifle below the maximal pressure that the first pulse-wave passes completely through under the cuff and causes the larger wave, upon which also a small shoulder is usually seen. Below this point the oscillations continue to increase in size and then begin to decrease, the manometer being watched

all the while. The point at which the oscillations are maximal is the *minimal* or *diastolic* blood-pressure. Sometimes, especially in arteriosclerotics, the oscillations decrease a little and then again increase at about 10 mm. lower pressure. In that case Erlanger has shown that the lower point or *second maximum* of oscillations is the one corresponding to the minimal pressure.

In order to keep these records permanent Fontaine marks off on the drum the points which correspond to each fall of 5 or 10 mm. Hg in the manometer. These marks are made by means of one

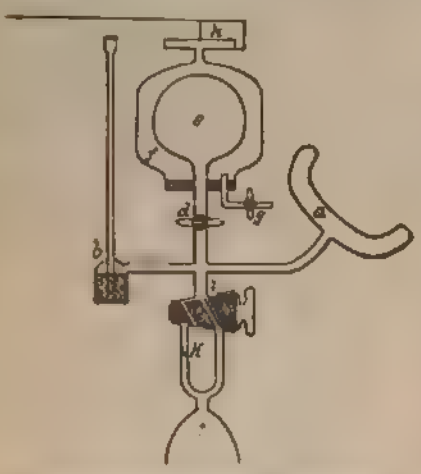

FIG. 23.—Diagram showing arrangement of Erlanger apparatus. After Howell.

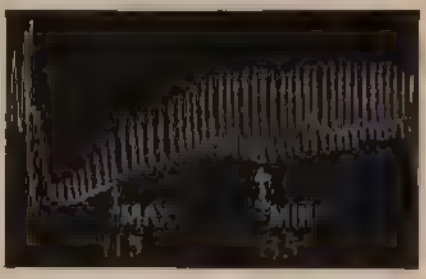

FIG. 24.—Curve taken with the Erlanger blood pressure apparatus, showing the points of maximal and minimal pressures.

of the tambours upon the polygraph (Hirschfelder) attachment which is caused to vibrate by squeezing a small pipette inserted into the long rubber tube.

This apparatus has been carefully tested, both upon animals and upon mechanical models, and has been shown to give accurate results. Of course it cannot be used unless the muscles of the arm are at rest, but neither can any other sphygmomanometer without introducing a large error. The readings obtained in determining both maximal and minimal pressures in arteriosclerotics are too high, but this error is also universal and at present unavoidable. At times the systolic increase in size is not sudden, but this can usually be remedied by pressing the lever a little more tightly than before against the drum. Occasionally a larger or smaller capillary outlet is required and these may be readjusted. The readings obtained by Erlanger's method are, as a rule, about 5 mm. higher for the maximal (systolic) pressure than by the broad cuff Riva-Rocci, and for the minimal (diastolic) within about 5 to 10 mm. of the readings by the method of Masing and Strasburger as modified by the writer (Hirschfelder, also Brush). Erlanger's method gives results sufficiently unequivocal to form the basis for a research, although the other method is often quite satisfactory.

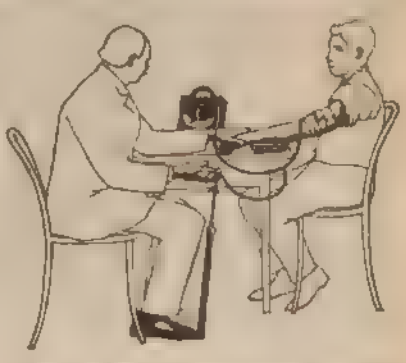

FIG. 25.—V. Recklinghausen apparatus. After v. Recklinghausen, *Arch. f. exper. Path. u. Pharmacol.*, lv.

V. Recklinghausen's Sphygmotonometer.—Numerous other sphygmomanometers have been devised of late, notably the *sphygmoscope* of Pal for visual determination of pressure by the movement of a drop of colored liquid, and the visual (tonometer) and graphic (tonograph) methods of v. Recklinghausen, upon the same principle as Erlanger's, but they do not possess any special advantages in their respective spheres of usefulness over the methods given above.

Auscultatory Method for Determination of the Blood-Pressures.—A very ingenious method for determining the maximal and minimal blood-pressures was devised by Korotkoff in 1905. If the pressure in a rubber cuff upon the upper arm is allowed to fall gradually from a point above the maximal arterial pressure, while the observer listens with a stethoscope pressed upon the brachial artery at a point about two centimeters below the lower border of the cuff, no sound will be heard until as soon as the pressure in the cuff falls below the maximal arterial pressure. As the minimal arterial pressure is approached, the second sound also becomes louder, reaches its maximum at the minimal arterial pressure (where there is the greatest alternate expansion and contraction of the artery), and disappears rapidly when the pressure in the cuff is a trifle below the minimal (diastolic) pressure. Indeed, Fellner has found this method accurate to within 5–10 mm. Hg of the readings with the v. Recklinghausen apparatus. Miss Allen and Mr. Eagle, in the study of the blood-pressures of 35 patients in the Johns Hopkins Hospital, found that the readings by this method never differed more than 2–5 mm. from control determinations made at the same time with the Erlanger apparatus, though they took the minimal pressure at the point at which the second sound absolutely disappeared.

The method seems therefore to be one of considerable accuracy, though in persons with very small vessels it may be difficult or impossible to use.

Pocket Form of Blood-Pressure Apparatus.—The exigencies of the busy practitioner demand an apparatus to occupy small space and yet give results of reasonable accuracy. To this end Potain made use of a small dial upon which the pressure was shown by the compression of a spring calibrated in centimeters of mercury. More recently a number of such forms have been devised in which the pressure chamber is connected with the usual Riva-Rocci-Recklinghausen cuff. The Tycos apparatus represents one of the most compact of these. It is practically a minature v. Recklinghausen sphygmotonometer, packed so small that it may be carried in the pocket with ease. The readings are made by the same method as upon the v. Recklinghausen, but the excursions are much smaller, a fact which often interferes with the determination of minimal pressure. Nevertheless, Messrs. Engle and Dandy, in the Johns Hopkins Medical Clinic, have found that determinations with this apparatus usually approach within 5–10 mm. of the determinations with the Erlanger apparatus. This error is usually due to the fact that the maximal pressure is determined by digital palpation, in which there is an inherent error of about this amount, regardless of the form of apparatus used. The determinations of minimal pressure usually fell within 5 mm. of those made with the Erlanger.

When the readings were made by the auscultatory method it was possible to reach absolute accuracy in many cases with this pocket form of apparatus. Like all spring pressure gauges, this spring is liable to wear out in time, so that it should be controlled every few months by comparison with a mercury manometer at various points throughout the range of pressure.

Gibson's Sphygmomanometer.—Still more recently Gibson and Sahli have devised an apparatus similar to Erlanger's but recording the oscillations of the mercury manometers directly by a float instead of by the Marey tambour. These instruments give results fairly concordant with the Erlanger and have the advantage of recording the corresponding pressure directly in absolute figures.

Normal Blood-pressures.—For young persons (19 to 25 years old) in the reclining posture the average blood-pressure according to Erlanger is maximal 110 mm., minimal 65 mm., pulse-pressure 45 mm. In general the limits in normal individuals at rest are maximal 110 to 135 mm., minimal 60 to 90 mm., pulse-pressure 30 to 45 mm. In the experience of the writer a maximal pressure of 115 to 120 mm., with a minimal of 75 to 85 mm., pulse-pressure 30 to 40, is more common.

MECHANISM OF THE CIRCULATION.

Pressure in Different Parts of the Vascular System.—Dawson has shown that the mean pressure is very constant throughout the arterial system while the maximal pressure falls greatly as one approaches the periphery. The minimal pressure is also quite constant. As one approaches the periphery the maximal pressure falls quite rapidly to meet the minimal, and in the smallest arteries they are practically equal. Hence the pressure

in these arterioles does not differ greatly from the minimal pressure in the aorta, although it is certainly a few millimetres less. The minimal arterial blood-pressure therefore represents the peripheral resistance (vasomotor changes), while the maximal pressure approximates the intraventricular pressure. Marey (l. c.) has shown that this approximation is closest when peripheral resistance is high. Accordingly the pulse-pressure, or difference between the two, represents the head of pressure tending to drive the blood from the heart through the aorta and large arteries onward into the peripheral arterioles. The fall in pressure may be compared to a cascade whose first descent is from heart to arterioles, whose second from arterioles to capillaries, and whose third is from the capillaries back to the heart. The actual head of pressure at any point in the arteries is never equal to the total head (maximal pressure) which would be active if the fall were uninterrupted by interposed resistance, but is more nearly equal to the pulse-pressure.

FACTORS DETERMINING MAXIMAL AND MINIMAL PRESSURE.

The fall in blood-pressure during diastole continues until the next systole takes place. If the pulse-rate is rapid the diastole is short and the blood-pressure has not time to fall much; hence, other things being equal, minimal pressure rises and pulse-pressure falls as pulse-rate increases.

FIG. 26. Diagram showing the maximal and minimal pressures in various parts of the circulatory system.

Erlanger and Hooker have claimed that under ordinary conditions the product of pulse-pressure multiplied by pulse-rate is tolerably constant, and represents roughly the velocity of blood flow, although Y. Henderson, the writer, and others have proved that this is very inaccurate and may involve an error of more than

50 per cent. The curves of Dawson and Gorham, who claim that the pulse-pressure is a "reliable index" of the systolic output (per beat) of the ventricles, indicate that these writers referred to qualitative rather than quantitative changes. Henderson has shown, however, that within a certain range of pulse-rate the ventricular output per beat varies inversely as the pulse-rate. Within this, the usual, range the velocity of blood flow is greatest. At rates below it time is lost during the periods of diastasis; above it the successive systoles encroach upon the period of ventricular filling and cut short the inflow. Within the limits indicated by Henderson, Erlanger and Hooker's index of velocity may often be correct, especially when there are no extreme vasomotor changes.

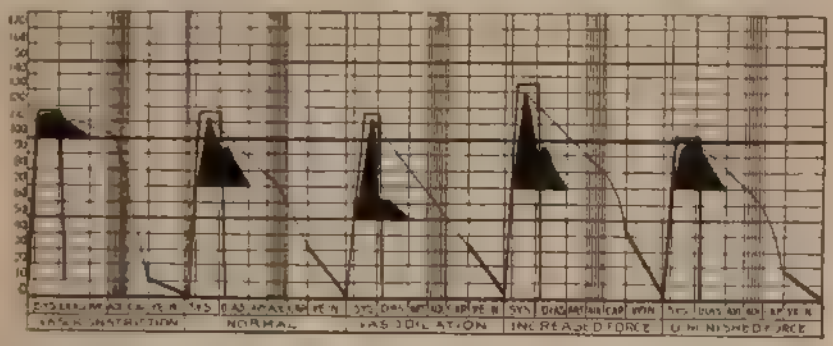

FIG. 27.—Diagram showing effects of vasoconstriction, vasodilation, increased and decreased force of ventricular contraction upon the maximal and minimal blood pressures and upon the form of the pulse. *SYS*, systole, *DIAS*, diastole, *ART*, large arteries, *AOL*, arterioles, *CAP* capillaries.

If the peripheral vessels dilate more blood can flow through in the same time, and hence when the pulse-rate is constant, vasodilation brings about fall in minimal pressure, rise in pulse-pressure; vasoconstriction brings about rise in minimal pressure, fall in pulse-pressure, but a change in maximal pressure following the change in minimal usually occurs reflexly. Figure 26 shows the various relations of maximal, minimal, and pulse-pressures to the state of the intraventricular pressure.

Erlanger and Hooker give the following table to indicate the conditions present in the circulatory system, but owing to the inaccuracy of the calculations this furnishes useful information only when the changes are extremely marked.

Maximal mean blood-pressure	Pulse-pressure × pulse-rate (velocity)	Energy of heart.	Peripheral resistance.
Constant	Increased	Increased	Diminished
	Diminished	Diminished	Increased
Increased	Unchanged	Increased	Increased
	Increased	Increased	Unchanged
	Diminished	Unchanged	Increased
Diminished	Unchanged	Diminished	Diminished
	Increased	Unchanged	Diminished
	Diminished	Increased	Unchanged

Changes in the peripheral vessels can be recorded by placing the patient's hand in a plethysmograph (Fig. 28) which presses hermetically about the forearm by means of a rubber cuff. The free space in the plethysmograph is filled with water which runs in or siphons out of a tube leading to a movable test-tube. Changes in volume of the arm are recorded by upward or downward movements of the test-tube.

Work of the Heart.—Since the intraventricular pressure is almost constant throughout systole, it is evident that the work done by the heart is tolerably constant throughout this period; and since no work is done during diastole, it is evident that the work of the heart per minute may be estimated, at least roughly, by the product of intraventricular pressure × duration of systole × pulse-rate.[1]

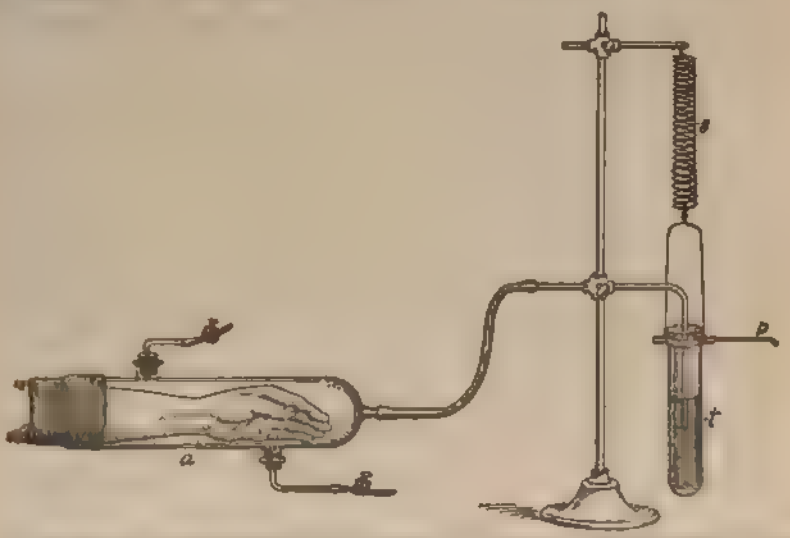

FIG. 28.—Mosso plethysmograph. (After Howell.) *a*, cylinder of plethysmograph; *t*, water or recording test-tube which moves by extension of spring *s* (spring *s* is arranged of a strength that keeps water in *t* at a constant level); *p*, point recording the excursions of test-tube *t*.

The value of blood-pressure determinations as an index of the functional power of the heart will be discussed on page 142 in relation to exercise and cardiac overstrain.

VARIATIONS IN BLOOD-PRESSURE UNDER PHYSIOLOGICAL CONDITIONS

1. Change of position.—Erlanger and Hooker have shown that the minimal pressure usually rises considerably and the pulse-pressure always decreases upon standing after having lain down. The pulse rate increases accordingly. They have shown that these effects are entirely due to the rule of gravity.

2. After meals the maximal pressure and pulse-pressure are increased, also the pulse-rate, and the minimal pressure may be increased but to a less extent. The circulation is accelerated.

3. After exercise the effect is the same as after meals, only more marked. When exercise is continued to the point of fatigue the pressures fall, the pulse-rate falls also, and the circulation is slowed (Schott, Masing, Cabot, Bowen). (See page 131.)

[1] For more complicated and perhaps more accurate formulæ cf. Tigerstedt (l. c.).

The ratio $\frac{\text{Pulse pressure}}{\text{Maximal systolic pressure}}$ or Blood-pressure coefficient is used more or less as an index of circulatory conditions. Its significance may be given as follows:

$$\left.\begin{array}{l} \text{P. P.} \times \text{P. Rate} = \text{Velocity} \\ \text{Syst. P.} \times \text{P. Rate} = \text{Work} \end{array}\right\} = \text{Efficiency of heart as a pump.}$$

In a normal individual this coefficient is 25 per cent. to 35 per cent.

4. Upon sensory stimulation the vasomotor centre in the medulla usually responds by constricting the peripheral vessels, and the pressure especially the minimal pressure rises. The pulse-rate usually quickens also. There are great variations in the response of different healthy individuals to pain sensations. Dr. A. Berg, under the writer's direction has tested the effect of pinching the ear upon the blood-pressure of healthy individuals and has found in some persons a rise of blood-pressure amounting to 10 to 20 mm. Hg, in others no effect, in others a fall of about 10 mm. Too intense stimuli produce shock. Mental exertion has a similar effect—a definite vasoconstriction setting in, which is shown by the shrinkage of the arm in a plethysmograph.

5. In sleep the opposite effects are seen, there is a general vasodilation and a fall in minimal blood-pressure (Howell, Brush, and Fayerweather). There is probably also a slight fall in maximal pressure.

VARIATIONS IN BLOOD-PRESSURE UNDER PATHOLOGICAL CONDITIONS

ASPHYXIA AND THE EFFECT OF EXCESS OF CO_2.

When the heart fails the circulation is slowed, and the blood becomes incompletely aerated and overloaded with CO_2 (f. Bohr). These conditions closely simulate the conditions present in asphyxia (Traube), or after breathing an atmosphere overladen with CO_2 (King has shown that the effect of these is quite similar).

Experimental Asphyxia. The conditions as observed in experimental asphyxia somewhat foreshadow those due to accumulation of CO_2 from heart failure. The blood-pressure changes in asphyxia have been most carefully studied by Konow and Stenbeck in Tigerstedt's laboratory, who found asphyxiation in rabbits resulting in the following series of events:

1. At the beginning of asphyxia the vasomotor and cardiac centres in the medulla are stimulated, as is also the inhibitory centre. Blood-pressure rises and the pulse is slowed. (Cameron has shown that, on the other hand, the tonicity of the heart muscle promptly decreases with the first stage of asphyxia and remains diminished throughout.)

2. As asphyxia continues the effect of slowing of the pulse exceeds that of the rise of pressure and the blood-pressure falls.

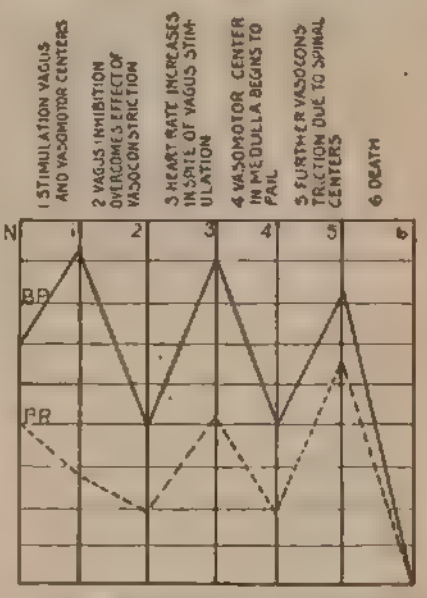

FIG. 39.—Diagram showing the curve of blood-pressure during asphyxia (Schenck, illustrating the results of Konow and Stenbeck). *N*, normal; *BP*, blood-pressure; *PR*, pulse-rate.

3. This condition slows the circulation still more, CO_2 accumulates in the blood, bathing the vasomotor centre, the latter stimulates the arterioles to still further constriction, the vagus can no longer overcome these effects, and in spite of its continued action the pulse quickens and blood-pressure again rises.

4. The activity of the vasomotor centre diminishes while the vagus centre remains at maximal activity, and the pulse-rate again slows and blood-pressure again falls.

5. The vagus centre fatigues, the accessory vasomotor centres in the spinal cord are again stimulated, and blood-pressure and pulse-rate again rise.

6. Conductivity of the heart diminishes, occasional beats are dropped by the ventricle, blood-pressure and pulse-rate fall, and the animal dies at this stage unless respiration is promptly restored.

Occasionally in asphyxia periodic changes in rhythm of the heart occur, such as have been described by Luciani in frogs and by Langendorff in cats. These irregularities occur when the vagi are sectioned as well as when they are active, this also occurs when the animal is made to breathe an excess of CO_2 (Klug). When, however, the vagi are inactive (cut), the rise of blood-pressure in asphyxia is continuous from the onset until the vasomotor centres fail (*i.e.*, in the fourth stage). When the cervical nerves have been cut and the vagi are active, there is an immediate fall in both blood-pressure and pulse-rate, the rise in blood-pressure sets in much later when the accessory vasomotor centres in the spinal cord are stimulated, or the animal may die if these fail to respond.

BLOOD-PRESSURE IN VARIOUS DISEASES.

Importance of Determining the Mechanism Producing the Change.—Variations in blood-pressure occur not only in conditions of health but still more under pathological conditions. As will be seen, the mechanism which brings these changes about is not always a simple one, and the causal factor may not be affected by merely resorting to therapeutic methods which lower a high blood-pressure or raise a low one. It is therefore necessary for the clinician to investigate as far as possible the condition of the vasomotor nerves, the strength of the heart-beat, to determine also whether the blood is properly aerated, and learn whether the kidneys are performing their function properly, before proceeding to symptomatic treatment of high or low blood-pressure when the cause is in any way obscure.

DISEASES WITH HIGH BLOOD-PRESSURE (HYPERTENSION).

The following represent the typical blood-pressure findings in various diseases. In exceptional cases more extreme variations are seen:

1. **Nephritis**, especially the chronic forms (maximal pressure 160 to 220, minimal 120 to 160, pulse-rate 50 to 80). High blood-pressure is common in both parenchymatous and interstitial cases. Passler and Heineke found that in animals from which almost all the kidney substance had been removed, blood-pressure rose *pari passu* with the occurrence of signs of renal insufficiency in the metabolism.

Excellent reviews of this subject have recently been published by T. C. Janeway and by Pearce. There seems to be a striking parallelism between continuous high blood-pressure and oversecretion of the adrenals, usually leading to an hypertrophy of the latter (see page 208).

In acute nephritis the blood-pressure may not arise, but Buttermann reports a case of scarlatinal nephritis where a rise of 50 mm. heralded the onset of the nephritis. Here it is of diagnostic and prognostic importance.

In uræmia blood pressure rises at the beginning of the attack, but may gradually fall a few days before a fatal termination (Luqueur). Gradual fall in blood-pressure also accompanies amelioration. Lugel finds that there is no rise in the mildest cases of nephritis, but that the rise of pressure runs parallel to the severity of the disease until the terminal fall sets in from cardiac weakness.

2. **Arteriosclerosis.**—Increased blood-pressure (maximal 150 to 170, minimal 110 to 130, pulse 60 or over) is the rule in arteriosclerosis, though there are occasional exceptions where the maximal pressure does not exceed or even reach 110 mm. (Israel). (See also chapter on Arteriosclerosis.)

with nitrites or other vasoconstrictors or by venesection only increases the task of the heart. The only medical treatment which aids it at all is administration of atropine to paralyze the vagi, quicken the heart, and permit the pressure to rise more readily. Lumbar puncture helps somewhat by removing the excess of intracranial fluid. If this does not suffice, Cushing advises surgical interference in many cases, a flap of the skull being lifted temporarily in order to relieve the intracranial tension and to allow the blood-pressure to fall. This procedure is almost devoid of danger in the hands of a surgeon whose asepsis is perfect, but very dangerous if it is imperfect, and this point alone will often decide the advisability or inadvisability of the operation.

7. **Attacks of Idiopathic Epilepsy** are associated with very high blood-pressure and slow pulse. The blood-pressure falls within a few minutes after the fit, which assists to differentiate it from uræmia (Pilez).

8. **Vascular Crises.**—Pal has described an important group of cases associated with crises of high blood-pressure due to vasoconstriction. Among these he classes uræmia, certain cases of arteriosclerosis, especially with abdominal and cardiac symptoms, and especially the tabetic visceral crises with intense pain. He has shown that these as well as attacks of lightning pains are associated with marked vasoconstriction and rise in blood-pressure, and states that they are even relieved by the administration of nitroglycerin. He also classes angina pectoris, intermittent claudication, and Raynaud's disease under this head.

9. **Attacks of Angina Pectoris.**

10. **Some Cases of Adams-Stokes Disease between Attacks.**—Gibson reports a case with maximal pressure 270, minimal pressure 70, pulse-rate 27. The pressure may, however, never rise materially. During the attacks it always falls almost to zero (see page 460).

11. **Exophthalmic Goitre** (Graves's or Basedow's disease) is often accompanied by hypertrophy of the heart with increased maximal, 140 to 160 mm., minimal 90 to 110 mm., and pulse-pressure 30 to 50 mm., pulse-rate accelerated to 120 and over. In some cases of Graves's disease the pressure remains low (maximal 120, minimal 90).

12. **The End of Pregnancy**, the onset of labor, and the puerperium are accompanied by a slight (10-15 mm.) rise of maximal pressure with little change in minimal pressure (Siemons and Goldsborough, see Part III, Chapter IX).

13. **Chronic Primary Polycythæmia.**—The increased number of red corpuscles increases the viscosity of the blood, and thereby the work of the heart, besides arteriosclerosis is usually associated. On the other hand, as shown by W. Erb, Jr., increase in blood-pressure causes liquid to leave the vessels and thereby increases the viscosity of the blood further—introducing a vicious cycle.

14. **Cyanosis in Heart Failure with Broken Compensation**, which occurs at some stage in almost all failing hearts. The blood becomes overloaded with CO_2, and vasoconstriction plus augmentation results as in asphyxia (see page 237). Usually the pulse is quickened, probably from fatigue of the vagus centre. This condition is of great clinical importance, since the high blood-pressure increases the work of the heart and accelerates its failure. Venesection, nitrites, digitalis, anything which accelerates the velocity of blood flow through the lungs, brings about improvement and lowering of the blood-pressure.

PATHOLOGICAL CONDITIONS ASSOCIATED WITH LOW BLOOD-PRESSURE (HYPOTENSION).

Although the occurrence of low blood-pressure is usually associated in the mind with the idea of a diseased heart, such is, as a rule, not the case. In fact, in most chronic diseases of the heart the maximal pressure is increased rather than decreased, as has been shown above. In one case of aortic insufficiency, for example, the writer found a maximal pressure of 150 and a minimal pressure of 110 two minutes before death, in spite of intense heart failure.

A low blood-pressure is more commonly an index of failure of the vasomotor centre than of the heart, and occurs in conditions where the strength of the heart is uninjured (Romberg and Passler, Husenfeld and Fenevessy, Crile).

Conditions in which low blood-pressure is found are:

1. **Acute infectious diseases**, except meningitis (where the blood-pressure is high from increased intracranial tension).—Romberg and Pässler have shown that bacterial poisons diminish the tone activity of the vasomotor centre, and may even paralyze it. The strength of the heart is shown to be undiminished if the vasodilatation is counteracted by adrenalin, compression of the abdominal aorta, etc. The blood-pressure falls because the arterioles are dilated and the outflow from the arteries is too rapid (maximal pressure 90 to 110, minimal pressure 50 to 90, pulse-rate increased, see table, page 29).

The lowest blood-pressure is in typhoid fever (Barach) and peritonitis, where the dilatation of abdominal vessels from the local inflammation add their effect to that of the cutaneous vasodilation. In typhoid fever the writer has seen maximal pressures as low as 65 mm. Hg (Riva-Rocci), although maximal 100 to 120 with minimal 60 to 90 is more common. Crile and Briggs have described rises in blood-pressure at the onset of perforation due to splanchnic stimulation, but the writer has had two cases (one of which is mentioned by Briggs) in which inactivity of the vasomotor centre prevented this rise from occurring.

In pneumonia the blood-pressure may not be changed much (maximal 110 to 130, minimal 90, pulse-rate 120); it may rise as mild asphyxia sets in, or it may fall very low from vasomotor paralysis.

In diphtheria, scarlet fever, measles, acute rheumatism, and and in fact in all other acute infectious diseases, the maximal pressure usually falls below 100 during the height of the fever (Weigert).

2. **Phthisis.**—In this disease all ranges of blood-pressure may be found. John, Naumann, Burckhardt, and Stanton have found uniformly low pressures, 90 to 100 mm. with the Gaertner and Riva-Rocci apparatus, but this may arise from the pallor of the skin. Janeway found that variations of maximal pressure between 80 and 120 mm. Hg are common in the same patient, and the writer's experience bears this out. The pulse-rate is usually rapid, 80 to 100 per minute. Peters finds that there is usually a rise of blood-pressure when improvement sets in, and a fall when the case is getting worse.

3. **Shock.**—Crile has shown that in surgical shock from injury or pain there is loss of tone activity of the vasomotor centre exactly as in acute infectious diseases. Syncope from emotional excitement, etc., is of similar origin. Crile counteracts the vasomotor paralysis by putting a double-walled rubber suit upon the patient and inflating the chamber between the walls until the pressure compensates for the loss of vascular tone.

Henderson, while confirming Crile's observations as regards the paresis of the vasomotor centre, believes that this is not the primary phenomenon. He calls attention to the similarity between the phenomena of shock and those of mountain sickness, which Mosso has shown to be due to a low CO_2 content of the blood (acapnia). Henderson believes that the mechanism of the two conditions is similar and is able to substantiate his claim by producing shock experimentally under all conditions in which CO_2 is made to escape rapidly from the blood, either through rapid aëration of the lungs or through exposure of the intestines and mesentery to a current of warm moist air. He has shown further that CO_2 is the hormone which preserves the tonic contraction of the walls of the veins. Bancroft has also shown that these are under nervous control as well as the arteries and that their nerves arise in the same regions as do the vasomotor nerves to the latter.

According to Henderson the sequence of events in shock is as follows:

(1) Pain or emotion, (2) hyperpnœa, (3) overaëration of the blood (lowering of the CO_2 content, acapnia), dilatation of the veins and accumulation of blood in the latter, lowering of the venous pressure (and hence diminished entry of blood into the heart); (4) fall in arterial pressure, accompanied by loss of arterial tone (vasodilatation), (5) cerebral anæmia.

There is an accumulation of blood in the veins with depletion of the arteries (an arterial anæmia).

In fevers the high temperature gives rise to a slight polypnœa and also favors the evaporation of CO_2 from the lungs. Henderson believes that these factors cooperate with the toxins in producing the vasomotor paresis of infectious diseases.

It will be seen that in many of the functional cardiac diseases accumulation of blood in the veins with depletion of the arteries is the most striking disturbance in the circulation, and plays an important rôle in producing the clinical picture.

Whether an occasional whiff of CO_2 or merely occasionally holding the breath will relieve the acapnia and restore the vascular tone in such cases remains to be proved.

4. **Collapse** from various poisons, carbolic and salicylic acid, arsenic, phosphorus, drugs of the antipyretic series, etc., is due to the same cause—failure of the vasoconstrictor centre, and likewise is accompanied by low blood-pressure.

5. After extensive **hemorrhage** a fall of blood-pressure sets in (except after venesection in some cases where a failing heart is relieved), owing to lack of blood to fill out the arteries. This is usually relieved by subcutaneous or intravenous NaCl infusion, or even by direct arterial transfusion (Crile).

6. In **diarrhœa, dysentery, cholera,** or after profuse vomiting, as from cancer of the stomach, intestinal obstruction, peritonitis, etc., when large amounts of fluid have left the body, the arteries may also be depleted of fluid and a very low blood-pressure result. This is also relieved by infusion.

7. In **pleurisy,** especially pleurisy with effusion, blood-pressure is uniformly low.

8. **Pericarditis** is accompanied by low blood-pressure (maximum 100 to 120, minimum 70 to 90, pulse-rate increased) unless complicated by hypertrophy of the heart or some other factors.

9. **Acute cardiac diseases** of all types, which have not been preceded by chronic processes and are not associated with marked cyanosis. Here the above-mentioned toxic action on the vasomotor centre is usually present if the endocarditis is of the infectious variety, and besides there is some weakening of the heart. The quickened pulse-rate prevents CO_2 from accumulating in the blood and the asphyxial rise in pressure does not occur. K. Weigert reports all ranges of pressure between 95 and 140 mm. Hg.

10. In **chronic mitral stenosis** the maximal and minimal pressures are usually normal or a little below normal, when the left ventricle does not hypertrophy, but this may vary considerably.

11. **Chronic wasting diseases,** cancer, chronic phthisis, anæmias, etc., are associated with brown atrophy of the heart muscle (see page 211), with weakened heart action, hence with lowered blood-pressure (10 to 20 mm. lower than normal, pulse-rate usually increased).

BLOOD-PRESSURE IN THE VEINS.

Various methods have been devised for the determination of the venous blood-pressure in man, the first being introduced by v. Basch and being but a slight variation of his arterial sphygmomanometer.

A very similar apparatus has been constructed recently by Sewall, but this gives rather unsatisfactory results in practise. V. Frey and later Gaertner also determined the pressure by considering it equal to the height above the angle of Ludwig at which the veins of the hand could be seen to collapse. This method is not quite as good as the former. A considerable advance was made by v. Recklinghausen, who compressed the vein by inflating a small rubber capsule provided with a glass window in the top and a rubber-dam floor having a hole in its centre. This dam was coated with glycerin so as to insure perfect contact. It is then placed over a vein, preferably upon the back of the hand or wrist, and the system blown up until the vein can be seen to disappear, at which point the pressure is read off upon a water manometer. Eyster and Hooker have modified this chamber by constructing one of aluminum with the entire top of glass and the two ends concave so as to avoid pressure upon the veins, and their apparatus seems to give results concordant within 1 cm. H_2O. They find that the normal venous pressure at the sterno-xiphoid articulation is 5–10 cm. H_2O.[1] It is increased by exercise and in cardiac cases with broken compensation, when it may rise to 27 cm. or over. When the veins are not sufficiently distended at that level the hand may be lowered a known distance, the pressure read, and the distance lowered subtracted from the amount of the reading will represent the venous pressure. In cases where phlebosclerosis is present no satisfactory determinations could be made.

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[1] These figures agree well with direct manometric determinations recently made in man by Moritz and v. Tabora (Verhandl. d. Kong. f. innere Med., 1909, xxvi, 378).

The pressure in the capillaries of vascular areas, especially of the lips, may be determined in the same way, using the point of blanching as the criterion.

The study of the venous pressure is of undoubted importance as an index of accumulation of the blood in the systemic circulation and thus as an index of heart failure. Moreover, it is the most important physiological factor bringing about variations in the volume of the heart; a high venous pressure causing dilatation, a low venous pressure causing diminution in volume (insufficient filling). This may prove to be an important factor in bringing about certain conditions in which there is "arterial anæmia" (shock, cardiac neurosis, etc.).

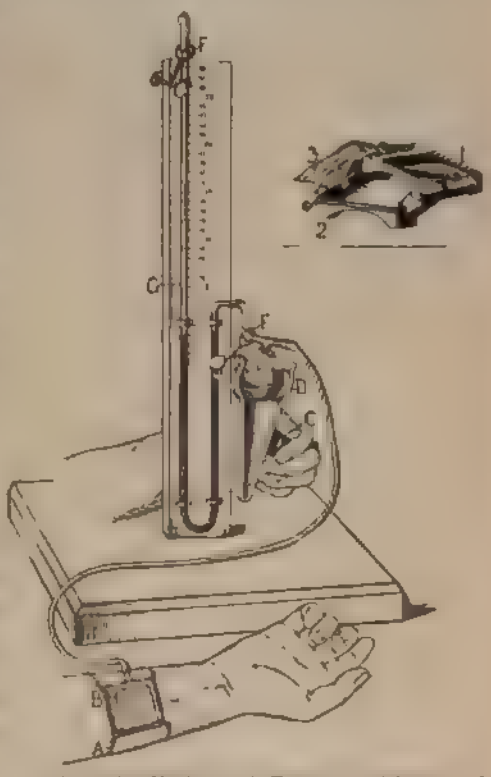

FIG. 31.—Hooker and Eyster's modification of v. Recklinghausen's method of determining the venous pressure in man.

THE PULMONARY CIRCULATION

Before birth the resistance in the vessels of the collapsed lung is greater than that in the systemic arteries, and hence blood passes from the pulmonary artery to the aorta through the ductus arteriosus (Botalli).

As the blood-pressure in young infants is 80 mm. Hg (Trumpp) it must be assumed that the pulmonary pressure is somewhat greater than this. When the area of lung capillaries widens with the first inspiration, the resistance in the pulmonary vessels decreases very markedly. This decrease continues during the period of infancy until the lung is fully expanded. According to a number of observers (Beutner, Lichtheim, Openchowski, Bradford and Dean, Plumner), the mean pressure in the pulmonary artery of rabbits, cats and dogs varies from 6 to 35 mm. Hg. It may be said to be approximately one-third that of the aorta but subject to considerable variations. Tigerstedt has found that in rabbits with 142 mm. pressure in the aorta the pressure within the right ventricle is much greater than this.

The pulse-pressure in the pulmonary artery is much smaller than that in the aorta, probably about 6–12.5 mm. Hg in small animals, and in man not far from these figures.

Work of the Right Heart.—The pressure within the pulmonary artery and hence the work of the right heart varies within wide limits under experimental conditions.

These variations are in part passive, due to passive stasis of blood within the pulmonary vessels, and in part may be the result of vasomotor changes in the pulmonary vessels.

The conditions in which the changes in pulmonary pressure arise passively from changes in the left ventricle are the most common and are clinically the most important.

Increased mean pulmonary pressure may arise

1. When an increased amount of blood enters the right heart from the veins and is expelled into the pulmonary artery.

2. The pulmonary blood-pressure also undergoes rhythmic variations, falling during inspiration as a result of suction (as shown by de Jager) and rising during expiration.

3. When the left ventricle fails to pump an equal amount onward into the aorta, causing blood to accumulate in the pulmonary capillaries until these are overfilled and aid in increasing the resistance in this circuit. (The left ventricle acts upon the pulmonary circulation as a suction pump.)

4. Probably from constriction of the pulmonary arteries under the influence of vasomotor nerves.

Pulmonary Vasomotor Nerves.—The existence of vasomotor nerves in the pulmonary artery, first suggested by Brown-Séquard (1870 to 1873) and later by Badoud, has been much disputed, but seems now to be proved.

François-Franck has shown that stimulation of the lower cervical and upper five thoracic ganglia in the dog uniformly caused a rise of blood-pressure in the pulmonary artery, a fall of pressure in the left auricle, and an increase in the volume of the lungs, probably due to accumulation of blood on the arterial side of the capillaries. This rise in pulmonary pressure bore no constant relation to the pressure in the femoral artery, which sometimes rose and sometimes fell. This evidence strongly favors the existence of vasoconstrictor fibres. François-Franck showed further that these same changes in pulmonary arterial pressure, left auricular pressure, and lung volume occurred reflexly when the central end of the femoral nerve or a proximal branch of the solar plexus was stimulated. This reflex, as he shows in a subsequent paper, may have important bearings in the production of certain cardiac symptoms and in influencing the course of cardiac diseases.

Action of Drugs on the Pulmonary Circulation.—François-Franck's researches are very convincing. They have been confirmed by H. C. Wood, Jr., and others, and are accepted by as keen a critic as Tigerstedt, but Wood, Jr., and also Petitjean have found that all drugs exert a much less marked effect on the pulmonary circulation than on the systemic. It must be admitted that acceptance is not universal. The clinical importance of the problem renders it a matter of universal interest.

It may be considered proved by François-Franck's work that sensory stimuli, stimulation of the sympathetic nerves, asphyxia, etc., may cause the pulmonary arterial pressure to rise to about double its original height, and hence in chronic conditions may play an important rôle in bringing about hypertrophy of the right ventricle. Moreover, changes of pressure which are relatively small when applied to the left ventricle assume much greater proportions when applied to the weaker right ventricle, and apparently slight changes in the strength of this chamber may then be important factors in the mechanism of the circulation.

Tonicity of the Right Ventricle.—More important than the changes in pressure in the pulmonary artery are the changes in tonus of the right ventricle. Owing to the thinness of the wall, changes in tonicity affect this chamber much more readily than they do the left. Overstretching of the fibres sets in more readily, and weakening of the right ventricle results more readily. These changes may have no direct relation to the changes in pulmonary arterial pressure.

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VISCOSITY OF THE BLOOD.

One of the most important factors in determining the work of the heart and the nutriment of the tissues is the viscosity of the blood, that is, the friction which its molecules exert upon each other and upon the walls of the blood-vessels.

Poisseuille and later Arrhenius introduced a method for determining viscosity quantitatively for indifferent fluids by observing the time taken for a given volume of fluid to flow vertically down a given length of capillary tube. The time taken by water to flow under the same conditions was used as the unit. Poisseuille found

Quantity of blood flowing in given time = viscosity coefficient × (diameter of capillary)⁴ × height of pressure : length of tube for distance of flow.

Huerthle found that Poisseuille's law also held for pulsating fluids and measured the viscosity of the blood in the living animal by comparing the outflow of blood from a capillary tube introduced into the aorta to the outflow of water under the same conditions.

$$\text{Viscosity coefficient (water)} = 4700$$
$$\text{Dog's blood} = 1045$$
$$\frac{\text{Water}}{\text{Dog's blood}} = \frac{4700}{1045} = 4.5 \quad \text{Coefficient of viscosity.}$$

Apparatus for Clinical Determination of Viscosity.—Various forms of apparatus have been devised for determining the viscosity clinically, most of them depending upon the time taken for a column of blood in a given capillary to traverse a given distance or to flow out of a given orifice when subjected to the pressure exerted by a constant column of water.

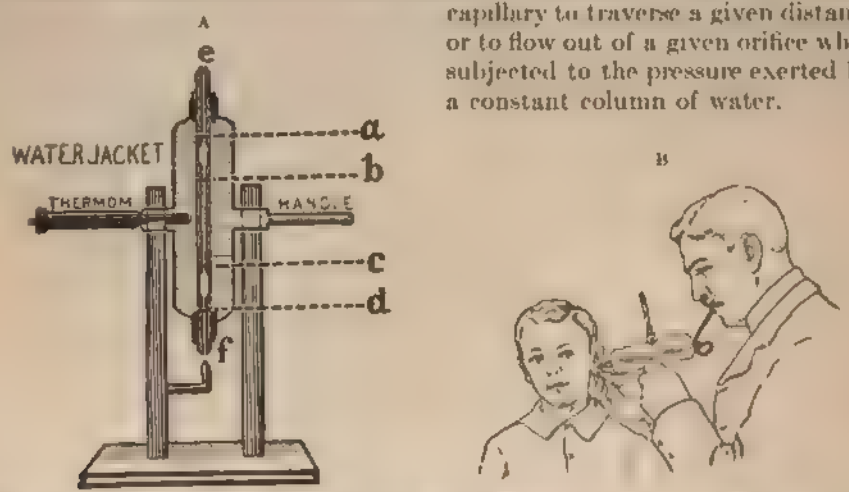

Fig. 32.—Determann's apparatus for determining the viscosity of the blood. After Brugsch and Schittenhelm. A, Apparatus at rest on its stand, pivoted on the thermometer and the handle as an axis. In the blood receiving tube within the water-jacket $ae = df$, $ab = cd$. B, Method of application. (After Brugsch and Schittenhelm.)

Such apparatus has been described by Huerthle, Burton-Opitz, Hirsch and Beck. Determann, Hess and also McCaskey have devised very simple forms of apparatus in which suction from a rubber bulb is used instead of positive pressure. C. R. Austrian in the Johns Hopkins Medical Clinic has found that the Hess apparatus gives results with normal blood which tally well with the blood count, and which therefore seem quite satisfactory.

Determann's newer apparatus (Fig. 32), however, combines clinical convenience with accuracy and is probably the most satisfactory now in use. It consists of a capillary tube surrounded by a small condenser-jacket of glass containing water at 38°. The jacket bears two side-arms which rest in the forks of two uprights so that the

jackets always assume a vertical position. The apparatus is taken up as a whole and the blood sucked up to a mark on the capillary. The apparatus is then placed back on the forks, and the time taken for the blood to flow out until it reaches a second (lower) mark is noted. (This should require 30–40 seconds.) A similar determination is made with water (6–8 seconds).

In order to keep the blood from clotting, a little hirudin may be placed upon the ear before stabbing it. This does not alter the viscosity as do adding sodium oxalate, laking, and defibrination, and keeps the blood from clotting for 20–30 minutes.

Determann obtains a few drops of blood quickly by having the patient exert a forced expiration with the glottis closed (Valsalva's experiment).

Factors Influencing Viscosity.—Heubner, Determann, and others have found that the chief factor in determining the viscosity is the viscosity of the red corpuscles, to which about two-thirds of the viscosity of the blood is due. Indeed in many cases these observers, and also Austrian, have found that the blood-count and the viscosity furnish accurate controls of one another, - though there are exceptions under pathological conditions (leukæmia, etc.). There is little if any difference between the viscosity of the normal blood in the arteries, capillaries, and veins. But in venous stasis the viscosity increases tremendously.

In a polycythæmica with 11,000,000 red corpuscles the viscosity may be three or more times the normal (Stern).

On the other hand, in anæmias, fever, the hydræmia which is associated with anasarca in broken compensation or exudates the viscosity is uniformly greatly diminished.

Burton-Opitz found that diet exerted a considerable effect, meat raising the viscosity, carbohydrates and fats lowering it. He also found that hot baths lowered viscosity while cold baths increased it. Hot-air baths seem to have little effect.

In compensated heart disease the water content of the blood does not change (Askanazy), nor does the viscosity, but the water is increased and the viscosity diminished (3.74 to 4.21) when compensation is broken (Determann). In bronchitis and diabetes it is high (5.5).

Determann cannot confirm the findings of Otfried Müller and Inada that potassium iodide lowers viscosity; and indeed the changes which they obtained were less than 1.0 per cent., well within the limits of experimental error. Their paper, as well as those of Hirsch and Beck, illustrates the tendency of workers in the field to draw too definite conclusions from too small variations.

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III.

THE ARTERIAL PULSE.

Historical.—Observation of the arterial pulse began almost synchronously with the accurate observation of disease in general. Hippocrates (B. C. 500) noted the marked pulsation (σφυγμός) of the arteries in certain diseases, but did not associate it with the beat of the heart. Herophilus (B. C. 300) observed the relative synchronism of these two events and speaks of the quiet pulse in health (σφυγμός) in contrast to the marked pulsation in disease (the τασμός of Hippocrates). Eristratus (B. C. 280) showed that the arteries near the heart beat before the arteries more distant from it. Aristotle and later Archigenes (first century after the Christian era) made numerous observations upon the pulse in various diseases, and the latter described and gave the name to the dicrotic type in cases of fever although he still believed that the arteries were filled with air. Galen (A. D. 131–202) demonstrated that the arteries were filled with blood and studied the influence of sex, age, climate, sleep, hot and cold baths upon the rhythm of the pulse.

The old Chinese physicians also described the pulse and even made drawings to illustrate their sensory impressions—a practice which did not begin in Europe until the time of Henri Fouquet in 1767. After Harvey's demonstration of the circulation of the blood (1628), the study of the pulse was resumed with renewed vigor and has continued to the present day.

Examination of the Pulse.—The characteristics of the pulse-wave are, as a rule, determined upon the radial artery, in which the arterial tension may be estimated as described on page 19, the wall of the artery being also rolled under the finger while the artery is empty, and thus the presence or absence of arteriosclerosis noted. The walls of a normal artery are barely, if at all, palpable, an atheromatous artery may feel like the trachea of a small animal (goose-neck), a diffusely sclerotic artery feels like a piece of thick-walled rubber tube.

It is important to note the palpability of several arteries, since one of them may escape a sclerotic process. All the blood must have been pressed out of their lumina and of the venæ comites that accompany them before palpation is begun, or else normal arteries may appear to be sclerotic. The pressure is then relieved, and the tips of two or three fingers are pressed upon the artery until the pulse appears maximal (at about the minimal pressure), when the following characteristics are noted: (1) whether the artery (hence the pulse) feels large and dilated (pulsus magnus) or small and constricted (pulsus parvus); (2) whether the pulse is hard (pulsus durus) or soft (pulsus mollis), *i.e.*, whether the minimal pressure is low or high; (3) whether the onset of the wave is sudden (pulsus celer) or gradual (pulsus tardus); (4) whether the wave is sustained (anacrotic) or subsides suddenly under the finger (collapsing, water-hammer, or Corrigan pulse); (5) the rate of the heart per minute (counted continuously during at least a half minute); (6) whether the rhythm is regular (pulsus regularis) or irregular (pulsus irregularis).

Clinical Sphygmographs.—An instrument (sphygmograph) to record the pulse-wave graphically was first devised by K. Vierordt (1855),

but it was not until 1860 that E. J. Marey devised a thoroughly practical and accurate form, almost devoid of error, which is still in use.

Marey's sphygmograph consists of a button (pelotte) pressed against the skin over the artery by means of a spring so as to receive the pulsations from the artery. It is held in place by a leather cuff and it is most important that the pelotte remain exactly over and not to one side of the artery. The pelotte is surmounted by a vertical rod or screw which articulates by a movable joint with a long writing lever. The writing lever records the magnified pulse movements upon a surface of smoked paper held in vertical position by a brass upright and driven by a small piece of clock-work.

A more compact and convenient form of sphygmograph is that of Dudgeon, in which the straight lever is supplanted by a double-jointed one which writes on a horizontal instead of a vertical strip of smoked paper. The tension of the spring pressing down the pelotte is roughly adjustable, which allows some variation in the pressure over the artery. V. Jaquet has improved Dudgeon's apparatus by adding to it a small time marker recording fifths of a second.

Another excellent form of sphygmograph is that devised by Roy and Adami, which, by means of a delicate adjustment, enables the observer to obtain a pulse record at exactly diastolic pressure. Unfortunately, it has never been placed on the market, and hence has not been subjected to the test of general use, but any one who is interested in sphygmographs should certainly familiarize himself with their observations.

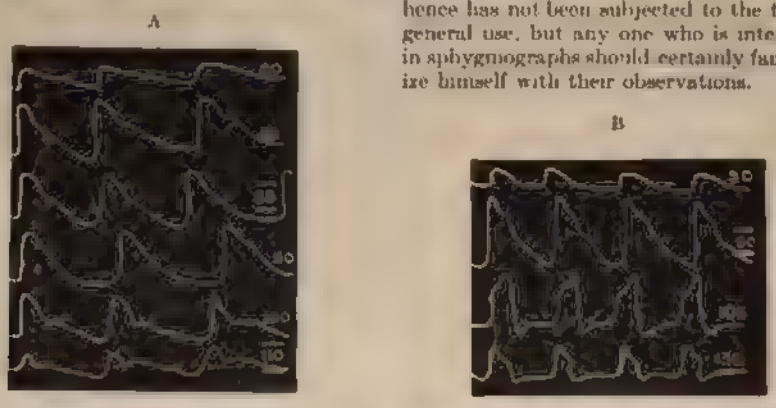

FIG. 33.—Brachial pulse-curves taken with the Erlanger blood pressure apparatus from the arms of two patients, merely varying the pressure in the cuff. The figures indicate the pressures at which the curves are taken, those underlined indicating maximal and minimal pressures respectively.

Errors in Sphygmography. In spite of the existence of these fairly satisfactory sphygmographs and of their wide use, discrepancies between the clinical observations and the tracings obtained are so great that Cabot refers to the sphygmograph as "an interesting little toy." The reason that it is not of value must be either that the apparatus itself is subject to inherent errors, or that, as Mackenzie states, "it was expected to give information of a kind that it was incapable of supplying." Unfortunately, both are the case.

Athanasiu, in investigating the accuracy of graphic recording devices, found that all sphygmographs which magnified the movement more than twenty times introduced a large inherent error; that of all the forms in use Marey's introduced the least error, while the Dudgeon apparatus and the Jaquet magnified it 130 times, introducing tremendous distortion from flinging large pulsations.

On the other hand, the writer, D. Gerhardt, and Stewart have been able to show that not only the size but also the entire type of the pulse-curve obtained depends upon the pressure exerted upon the artery and other similar factors, the true form of the pulse-wave being obtained only when the pressure exerted by the sphygmograph is exactly equal

to the pressure within the artery. Fortunately, this is the point at which the pulse excursion is maximal, and as all observers strive for the largest excursion, it is probable that most sphygmographic records are taken at about this pressure. The ideal apparatus is the one in which it is not merely probable but certain, and hence that of Roy and Adami is the only one which absolutely fulfils the requirements.

FIG. 34.—Absolute sphygmograms all of which correspond to the radial tracing above. The figures to the left indicate pressures in mm. Hg.

The Absolute Sphygmogram.—A very convenient and instructive method of recording pulse tracings has been introduced by Sahli. Sahli transfers the pulse-curve to coördinate paper upon which the ordinates represent millimetres of mercury and the abscissæ represent fractions of a second. The lowest point of the pulse-curve he marks at the level corresponding to the minimal blood-pressure, determined at the time with the sphygmomanometer; the highest point at the level corresponding to the maximal pressure; and maps out besides this the other main points of the pulse-curve (predicrotic fall and wave, dicrotic notch, summit of dicrotic wave, etc.) at heights and distances proportional to their occurrence upon the sphygmogram, but translated to this new scale of pressure and time. This curve he terms the absolute sphygmogram.

The absolute sphygmogram can also be read off from the ordinary sphygmogram by using the lowest point on the tracing as the ordinate of minimal pressure and as a base line for determining the pressure at other points, and calculating these from the proportion

Ordinate of point : Total height of pulse-wave =
Pressure at that instant (above minimal arterial pressure) : Pulse-pressure.

Discrepancies between Feeling and Recording the Pulse.—Not all the discrepancies between sensory impression and sphygmogram are the fault of the instrument. In the first place, there is no absolute uniformity in the minds of physicians as the standard to be applied to the individual pulse. Thus, the writer has seen one eminent clinician dictate a note, "pulse not collapsing," and another a few minutes later state that the same "pulse is collapsing in quality." The pulse had not changed, but the subjective criteria of the two men were slightly different.

Again, between pulse palpation and sphygmogram there is a difference. It is very difficult, almost impossible, to determine just how long a pulse is sustained and how quickly it falls, since these judgments are based upon a sequence of events lasting for an interval of about one-tenth of a second, and changes both in time and in pressure must be considered without the presence of any simultaneous standard for comparison. Psychologically, such comparisons must be very fallible. Practically they are not as fallible as they appear, for the judgment is based not upon form or duration, unless the abnormalities are marked, as much as upon changes of pressure. What one really appreciates most in feeling the pulse is the amount of maximal pressure ("hardness" of the pulse) and the amount of the pulse-pressure (size of pulse), and only to a lesser extent the duration of the pulse-wave. Hence, the sensation due to a high pulse-pressure with a moderate diastolic pressure is often mistaken for that due to a collapsing pulse, though the form of the pulse-

wave may show that it is quite well sustained. In comparing the pulse sensation with the sphygmogram, one is therefore comparing two somewhat different standards, and this inherent difference must be taken into account.

Significance of the Pulse-curve.—Assuming, however, that one has obtained a correct tracing from the artery, what deductions are allowable? It is evident that the artery expands somewhat under an increase in pressure (causing a rise in the pulse-wave) and contracts when pressure decreases (causing a fall in the pulse-wave). Further, the pressure in the artery increases or decreases, depending upon whether more blood enters it than can leave it at that instant (Fig. 35, $I > O$) or whether the reverse is the case ($I < O$). When the inflow exactly equals the outflow ($I = O$), no change of pressure occurs and a plateau results. The pulse tracing is merely the record of these events—the record of the ratio that the inflow into the artery from the heart bears to the outflow toward the periphery at each instant of the cardiac cycle.

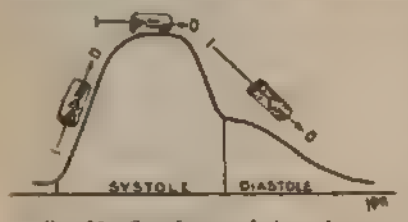

FIG. 35.—Significance of the pulse-curve. *I*, inflow into the artery from heart. *O*, outflow from the artery toward the periphery.

The normal pulse-wave has the following forms: an upstroke more or less steep (percussion wave), a rather acute summit, and sudden fall (predicrotic) followed by a very small rebounding wave (predicrotic wave), then another more gradual fall terminating in a small notch (dicrotic notch) which marks the end of systole (Marey, Huerthle), then a gradual fall during diastole. In the aorta the fall in waves is not as steep as in the radial artery, which indicates that the former reflects the conditions near the heart, the latter shows the conditions at the periphery (Marey).

FIG. 36.—Diagram showing the time relations of ventricular volume and pressure curves to pulse tracings from the aorta, carotid and radial arteries. Time divisions in one-tenth seconds. (Schematic.) Dotted lines represent curves taken with high peripheral resistance.

Relation of Pulse Form to Peripheral Resistance. There are three general types of pulse (Marey, Hirschfelder) which may occur without any heart lesion whatever, and even in the same individual at the same maximal and minimal pressures, though usually the maximal and minimal pressures vary with these conditions. (Fig. 37.)

Type I corresponds to marked peripheral dilatation, as after exercise, after meals, in shock, fevers, or in some nervous individuals with vasomotor instability. This is the collapsing type of pulse, rapid rise and rapid fall sometimes followed by a large dicrotic wave (see page 45). The rise is, however, about two hundredths of a second slower than normal, but this difference is not within the limits of perception. It

feels more sudden because it is sharply followed by the sudden fall. The fall in this type of pulse is almost complete before the end of systole, *i.e.*, before the dicrotic notch which marks that point (Marey, Huerthle).

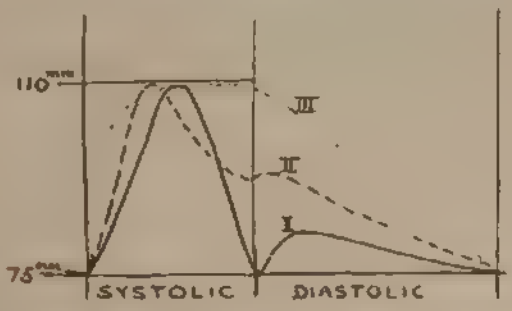

FIG. 37.—Three types of arterial pulse-curve corresponding to the same pulse-pressure and same pulse-rate. Johns Hopkins Hosp. Bull. xviii. I, vasodilation; II, normal; III, vasoconstriction.

In Type II only about half the fall occurs during systole. This corresponds to moderate degree of dilatation and is the type present in normal individuals.

In Type III the wave soon rises to the summit and remains there, forming a sustained plateau (outflow = inflow) until the end of systole, when it gradually falls. This corresponds to peripheral constriction, preventing the outflow from the aorta from exceeding the inflow into it, as is the case where a normal degree of dilatation is present. The normal pulse in man may be converted into this type by compression of both femoral arteries (Marey), or of the abdominal aorta (Stewart). The mere increase of the blood-pressure is not a cause, because after exercise the blood-pressure is increased and yet the pulse becomes more collapsing than before.

FIG. 38.—Effect of inhalation of amyl nitrite upon the pulse-form. After v. Kries. Curves taken in succession. Vasodilation reaches its maximum at a and diminishes at d and e. Well marked dicrotism at f, β and α indicate secondary waves due to elasticity of the artery.

These general outlines of the pulse waves are further modified by smaller wavelets due to the elastic vibrations of the artery wall or to the rebound of the percussion wave at the periphery (v. Kries). The most important of these is the dicrotic wave following immediately upon the closure of the aortic valves and due either to a centrifugal wave from the blood impinging against them, or to a reflected centripetal wave from the periphery toward the heart (v. Kries). Whichever theory may be correct, the essential fact remains that the dicrotic wave is a secondary one and is dependent upon arterial elasticity. V. Kries has shown that the dicrotic wave is most marked when the peripheral vessels are considerably dilated, but not when they are dilated to their fullest extent (Fig. 38).

The other waves may occur upon either upstroke (anacrotic) or upon the downstroke (katacrotic, Fig. 38, α, β) and are designated accordingly. Small secondary waves of this

type are most marked when the pressure is high and the heart action strong (*e.g.*, pulsus bisferiens), but their occurrence is often due to twitching of the tendons near the pelotte of the sphygmograph, and too great weight must not be attached to them.

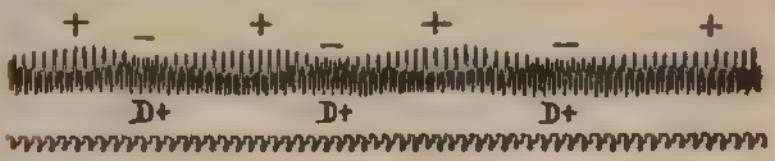

FIG. 39.—Mercury manometer tracing from the carotid artery of a dog, showing rhythmic variations in blood-pressure and rhythmic increase in dicrotism. (Kindness of Prof. Abel and Dr. Rowntree.) The dicrotic wave increases at the points *D* (+) at which the blood-pressure is lowest (−) and the peripheral arteries are dilated. Time in seconds.

Too much information should not be sought from the sphygmogram. All that should be looked for is whether the upstroke is sudden (p. celer) or gradual (p. tardus); whether the main fall in the wave begins early or late in systole, or not until the beginning of diastole; also whether the fall is quite or nearly complete before the end of systole. All possible mental reservations should be made for fling of the lever, incorrect applications of sphygmograph, etc., before a judgment is made.

FIG. 40.—Diagram showing various forms of pulse-curve encountered in health. Systolic portions of the curve are underlined. *HYPERDICROT*, hyperdicrotic.

THE PULSE-RATE.

The normal pulse varies considerably in different individuals, being in general more rapid in those of small stature and slower in persons of larger stature, hence, more rapid in women than in men. It also varies considerably according to age, being dependent upon the relative tone of vagi and accelerators. The pulse-rate is also more rapid (tachycardia) in fevers, varying in general according to the temperature—each degree Fahrenheit increase corresponds to an acceleration of about four to five beats per minute. C. D. Snyder, as the result of a long series of experiments upon the heart-rate in different vertebrates, finds that the rate is influenced by temperature in the same degree as is the velocity of simple chemical reactions and follows the logarithmic formula

$$2_{10} = \frac{K_1}{K_0}\left(\frac{10}{t_1 - t_0}\right)$$

In typhoid fever there is often an exception: a temperature of 103° to 105° being accompanied by a pulse-rate of about 90 per minute, owing to a toxic stimulation of the

TYPES OF PULSE IN VARIOUS DISEASES.

The following types of pulse are associated with various pathological conditions and corresponding states of the heart and vessels.

Type of pulse.[1]	Shown in Fig.	Characteristics.	Clinical conditions in which it is most frequently observed	Blood-pressure associated with it.			Vascular condition.
				Maximum.	Minimum	Pulse-pressure.	
Normal....	40	Sudden rise, sharp apex, slight predicrotic fall; then slow fall, small dicrotic wave, gradual fall in diastole	Normal individuals	Normal ..	... .	.....	Normal.
			Some cases of aortic insufficiency	High.....	Normal...	Increased.	Dilated.
			A few cases of fever	Normal or diminished	.....	Pulse-rate quickened	Dilated.
Anacrotic.	40	Sudden rise or slightly rounded plateau top lasting almost to dicrotic notch which is small, gradual diastolic fall	Arteriosclerosis; chronic nephritis	High	High....	Slightly increased or unchanged	Vasoconstriction.
			Some cases of aortic insufficiency				
			Some normal individuals	Normal...	High.....	Slightly diminished	Vasoconstriction.
Bisferiens.	40	Resembling anacrotic except that the small predicrotic fall is followed by rise equal or above that of the percussion wave, making the summit bifurcate	Arteriosclerosis; chronic nephritis	High.....	High.....	Increased	Vasoconstriction.
			Hypertrophied heart acting strongly				
Tardus....	40	Gradual slow rise, percussion wave oblique, summit round, gradual fall	Aortic stenosis ..	Slightly or greatly elevated	Elevated..	Increased or normal	Vasoconstriction.
Collapsing.	40	Steep rise, apex sharp, sudden steep fall, dicrotic notch in lower half of curve often level after the predicrotic wave	Aortic insufficiency (water-hammer or Corrigan pulse)	.....	Low or normal	Increased	Vasodilatation.
			Fevers	Normal or low	Normal or low	Normal or increased	Vasodilatation.
			Normal individuals, neurasthenics	Normal or low	Normal or low	Normal or increased	Vasodilatation.
			Some cases of Basedow's disease	Increased	Slightly increased	Increased.	Vasodilatation.
Dicrotic...	40	Collapsing in quality but dicrotic wave very pronounced and palpable, as a small wave regularly following soon after the percussion wave	Fevers, especially typhoid	Normal or subnormal	Normal or subnormal	Normal or increased	Vasodilatation.
			Normal individuals during or after exercise	Increased	Normal or increased	Increased	Vasodilatation.
			Neurasthenics, after amyl nitrite or nitroglycerin	Normal or increased	Normal...	Increased	Vasodilatation.
Hyperdicrotic	40	Dicrotic wave occurs at the foot of the ascending instead of descending limb	Any of the conditions in which dicrotism may occur, but with more rapid pulse-rate.	Normal or increased	Normal...	Increased	Vasodilatation.

[1] For forms of irregular pulse see page 63.

vagus; while in meningitis the high intracranial pressure may bring the rate down to a great deal lower (50 to 60) and may cause irregularity. In tuberculosis the pulse is rapid even in the early stages. The pulse-rate is also accelerated in the anæmias, in neurasthenia, Graves's disease, hysteria, shock and collapse, abdominal distention, peritonitis and other diseases of the abdominal viscera, and in numerous cardiac diseases. In fevers and in many other conditions of acceleration the pulse becomes extremely small and barely palpable on the one hand, and extremely rapid, barely countable on the other—a small and "running" pulse. Pulse-rates of over 160 per minute are not uncommon in fevers, while 200 or even 300 is reached in paroxysmal tachycardia. At these great rates the duration of systole is markedly shortened, as well as that of diastole (the period of systolic output falling from 0.26 sec. to 0.2 or even less).

Slow pulse (bradycardia) (below 60 per minute) is observed especially in conditions with intracranial tension in meningitis, in digitalis poisoning, chronic nephritis, chronic myocarditis, in convalescence from some fevers, especially diphtheria and influenza, and in Adams-Stokes disease. In the latter condition the auricles and ventricles are beating independently (see chapter on Adams-Stokes disease).

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IV.

THE VENOUS PULSE AND ELECTROCARDIOGRAM IN HEALTH AND DISEASE.

THE NORMAL VENOUS PULSE.

As has been seen, the study of the blood-pressure and of the arterial pulse conveys information regarding the strength of the heart-beat, the condition of the peripheral arteries, and the velocity which the heart is imparting to the blood stream. But it reveals the action of the left ventricle only, and what occurs in the other chambers of the heart must be sought for elsewhere.

In studying the heart from the four stand-points of Engelmann, rhythmicity, irritability, conductivity, and contractility, it is necessary to obtain a knowledge of the origination of the impulses in or above the right auricle (atrium), of whether impulses other than those causing the normal rhythm are acting upon that chamber, of whether the right auricle (atrium) is itself contracting, and of whether all the impulses are being properly conducted to the ventricle. Our knowledge upon these points has been derived almost entirely from the study of the pulsation in the jugular vein.

Visible Pulsation in the Veins.—Pulsation over the veins is visible in 80 per cent. of healthy individuals (Hewlett) and is as pronounced as that over the arteries, but it is different in character. The latter shows the force-pump, the former the suction-pump action of the heart. The pulsation over the arteries is quick, sharply localized, easily palpable, and the impulse is more marked than the collapse; that over the veins is diffuse, wavy, rarely palpable, and the collapse is more marked than the impulse itself. Further, the pulsations over the vein under normal conditions are exactly twice the number of those seen over the artery, and the first of the collapses is synchronous with the impact in the artery. Such a pulsation over the vein is known as the "physiological," "negative," or "double" venous pulse, in contradistinction to the other types of venous pulse to be described later.

The pulsation over the veins is not, like the arterial pulse, to be seen in every vein in the body, though Morrow has shown that in dogs it can be detected by means of delicate manometers. To the eye and to the recording apparatus available upon man, it is appreciable only in the veins near the heart, the external and internal jugular, the cephalic, and the axillary. Occasionally it is also to be seen in the brachiocephalic and other veins in the arm.[1] The site where it is most easily and uniformly seen is in the right supraclavicular fossa, either over or just to the right of

[1] Friedreich thought that this pulsation was transmitted from the arteries through the capillaries to the veins, but such transmission probably never takes place and other explanations must be sought.

the origin of the sternocleidomastoid. Sometimes it is a little more marked in the supraclavicular fossa at about the mammillary line where the external jugular vein enters the subclavian. The normal venous pulsation is rarely to be seen when the subject is standing or when propped up high upon pillows, but is most distinct after he has been in reclining posture for some minutes with a single pillow under his head and neck. In patients with venous stasis, on the other hand, it may be necessary for the patient to sit upright before any undulations appear.

It must be borne in mind that the pulsation seen and recorded over the veins represents the alternate filling and collapse of the latter. The collapse, that is the obliteration of the lumen of the vein by the atmospheric pressure, is usually the most important factor. It is evident that a wave will occur during those periods in which the pressure within the vein is greater than the atmospheric, and a collapse will occur whenever it is less. If it is permanently less (negative), the vein will remain collapsed; if it is permanently a little greater, the vein will remain distended. In neither case will a pulsation be seen.

The normal pulsation is best seen when the pressure in the jugular vein is alternating between a positive and a negative pressure during the different phases of the cardiac cycle. The elastic distention of the vein is not called into play. The elastic distention of the vein at systole occurs only at a much higher venous pressure, as in tricuspid insufficiency. Occasionally, especially in chronic heart cases with phlebosclerosis, the veins stand out like large knotty cords, but no pulsation is to be discerned in them at all. The knotty appearance (Fig. 11) is due to the closure of the valves within the veins, the dilatations appearing just above the valves. Perhaps the closure of the valves prevents or dampens the pulsation, or perhaps the rigidity of the vessel wall prevents it from collapsing and filling. Normally the valves in the jugular do not close, but this closure is brought about by chronic venous stasis, just as it is in quadrupeds where back pressure results from the head being dependent. In such cases it is impossible to obtain any idea of the undulations nearer the heart.

GRAPHIC RECORDS OF VENOUS PULSATIONS.

A far more exact idea of the nature of the jugular pulsation can be obtained by recording it graphically than by mere inspection. With proper apparatus this is not accompanied by any difficulty, and a satisfactory record of both venous and carotid pulsations can be obtained in about the same time as a radial sphygmogram. For the interpretation of the venous tracing it is necessary to compare it with the other events of the cardiac cycle, which is accomplished by using the pulse-wave from some artery to fix the standard of time.

In order to interpret the waves upon the venous pulse, it is necessary to record simultaneously the venous pulse and either the arterial pulse or the cardiogram, and to see at which point in the cardiac cycle each event will fall. Accordingly, all forms of apparatus (polygraph) for obtaining such records are arranged for taking at least two records simultaneously. In all of these the pulsation from over the vein is received in the same way, and the only difference in the various forms of polygraph lies in the method of obtaining the arterial tracing and in the form of kymograph used.

Application of the Receivers.—The pulsation in the jugular vein is recorded by holding over the skin above it a small glass funnel or special receiver (Fig. 11, c), which is connected with a Marey recording kymograph tambour. The movements of the skin are

transmitted at once to the kymograph tambour and recorded by the lever. As a rule, the most favorable conditions are obtained when the patient is lying with head and neck supported on a single pillow that extends down just to the shoulders, with his head turned well to the right and the neck definitely flexed. In this way the right sternocleidomastoid is relaxed and a tracing over the pulsation from base of the internal jugular vein is transmitted to the skin. When this is not obtainable the junction of the external jugular vein with the subclavian should be tried in the same way. The funnel should be pressed against the skin just enough to make the contact air-tight without affecting the pulsation, but this is effected without any great dexterity and oscillations due to the holding of the receiver rarely appear upon the tracing. When they do so it is in the form of fine oscillations bearing no relation to the cardiac cycle and having a rate of from four to eight per second, in contrast to the much slower and larger movements in the veins. Such tracings should be discarded.

In many cases the simple glass funnel is not as satisfactory as a receiving device introduced by Mackenzie (Fig. 41, c), consisting of a shallow metal pan 3 cm. in diameter with a tube leading off from it in the form shown in Fig. 41, one portion of the circumference being flattened instead of round in order to fit closely above the clavicle. It is convenient to have a small hole in the top of the pan so that it may be adjusted to the skin without moving the recording lever, and after adjustment is complete the hole is closed by placing the finger over it.

The tracing from the carotid artery is obtained in a similar way, using for a receiver a small tambour surmounted by a button to fit over the artery (Fig. 41, b). A small hole in the top of this tambour serves the same purpose as before and is also stopped by covering with the finger. The carotid artery is next to the skin just inside the sternocleidomastoid when the head is turned toward the corresponding side, the pulsation being most marked when the receiver is pressed heavily upon it.

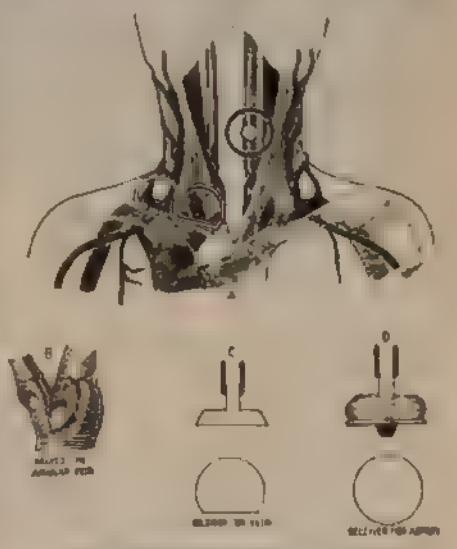

FIG. 41.—Sites for recording the jugular and carotid pulsations. *A*, distribution of the veins shaded in black, showing the sites for applying the jugular receiver truncated and the carotid receiver concentric circles; *B*, appearance of the valves within the jugular vein when closed by back pressure; *C*, receiver for jugular vein; *D*, spring tambour for recording the pulsation over the carotid artery.

Comparison of Carotid and Jugular Pulsation.—Since the jugular vein and the carotid artery are at about the same distance from the heart, the tracings from the latter must always be compared with the former in order to exclude waves which might have been transmitted to it from the artery, and also to indicate the relations of the venous waves to the cardiac cycle.[1]

This comparison may be made by taking the jugular and the carotid tracings simultaneously and comparing them with each other directly,[2] or, for the sake of convenience,

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[1] Where great accuracy is necessary the onset of the *c* wave must be compared with that of the apex beat.

[2] It is not necessary that the levers be exactly superposed, but it is preferable to measure off the distance of the given point horizontally from the arc described by the lever at the beginning of the tracing (*e.g.* Fig. 44). This distance is then laid off upon the other curve in the same manner. Wherever the curve may begin the paper traverses the same distance upon both curves in the same time.

a carotid and a brachial or radial tracing may be made simultaneously and the point at which the carotid wave begins marked off upon the latter. Then a jugular and a brachial tracing may be made, and the time that the carotid wave occurs before the brachial marked off before each brachial wave in this tracing, and these points then measured off upon the jugular tracing. This is often the simplest and quickest procedure.

Respiration Recorder.—It is often of importance to determine the relation of an arrhythmia to the phases of respiration. The simplest device for recording the latter consists of a piece of rubber tube (Fig. 42, RUB) connected with the tube to the recording tambours by a short L-shaped piece of glass tubing (GL). A piece of string or tape is attached to the rubber tube, another to the glass tube. The apparatus is then put on so as to encircle the level of the nipples. The strings are tied tightly enough to just stretch the rubber tube during expiration. Inspiration then causes a downstroke of the levers, expiration an upstroke.

FIG. 42.—Apparatus for recording the respiration. *RUB*, rubber tube, *GL* glass tube.

Forms of Polygraph.—Several forms of polygraph for clinical purposes have been devised to record these curves. Their relative value depends largely upon the delicacy of the tambours. The oldest form is the polygraph of Marey, consisting of an ordinary kymograph drum arranged to rotate horizontally with two Marey tambours to write upon it, so as to record simultaneously the curve from the jugular and carotid or jugular and cardiogram. This is fairly satisfactory, but in mechanical perfection some others are superior. Mackenzie has devised two forms of polygraph. The first, a simple Jaquet sphygmograph upon which a Marey tambour is mounted in addition so as to record the radial pulse and jugular or carotid, etc., simultaneously, the time being marked off in ¼ seconds by a small clock-work as well. In the improved form of Mackenzie polygraph, the levers bear ink pens and write upon an endless roll of white paper, so that a very long series can be obtained. V. Jaquet's cardiosphygmograph differs from the simple sphygmograph only in bearing in addition two Marey tambours whose double-jointed levers write just above the lever attached to the radial pelotte. Both Mackenzie's and Jaquet's methods suffer from the inconveniences of adjusting the sphygmograph to the radial artery and keeping it adjusted during the entire observation, a factor which is very disconcerting to both patient and physician and which prevents many important observations from being taken on restless patients.

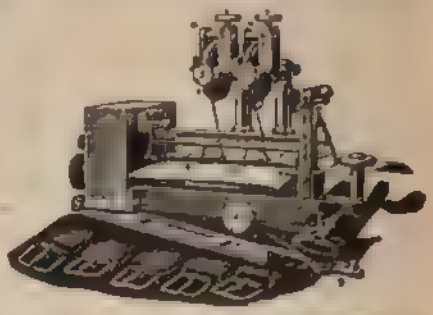

FIG. 43.—V. Jaquet's cardiosphygmograph. Kindness of A. H. Thomas Co.). *a*, time-marker (¼ sec.), *b*, *c* levers of tambours for recording venous tracing, carotid pulse or cardiogram; *d*, lever recording radial pulse-wave.

This difficulty is obviated in the writer's modification of the Erlanger blood-pressure apparatus (Fig. 22, page 21), in which two small Marey tambours and a time-marker are arranged to write above the lever of the blood-pressure apparatus. When the bag is inflated upon the arm, the brachial pulse is recorded by the lever of the blood-pressure apparatus and used as the standard instead of the radial pulse. This entails no trouble and no expenditure of time, thereby saving much of the trouble given by the other methods, and permits a set of records to be obtained very

quickly. It is also possible for the operator to work with one hand free and thus save the necessity of an assistant. The curve thus obtained from the jugular vein is shown in Fig. 44 and its relation to the other events in the cardiac cycle shown in Fig. 45.

FIG. 44.—Normal venous tracings. *a*, wave due to auricular contraction; *c*, wave at onset of ventricular contraction, the vertical line *c* representing the beginning of the carotid pulse-wave; *x*, the bottom of the mesosystolic collapse; *d*, time of dicrotic notch in the carotid; *v*, wave at end of systole; *y*, hollow at the end of the postsystolic collapse; *t*, *d* (*Bard*), telesystolic and protodiastolic waves described by Bard. (The *x* and *y* depressions are not lettered on all tracings.)

Recently, Uskoff has constructed a very compact form of this apparatus, bearing an Erlanger blood-pressure apparatus, a tambour for recording the height of the blood-pressure objectively, a tambour for apex or venous or carotid tracings, and an excellent time-marker. This seems to be a very good instrument of wide applicability suitable to all the needs of the practitioner.

The choice of apparatus depends chiefly upon the delicacy of the tambours and upon the portableness of the apparatus. In the latter regard the Jaquet cardiosphygmograph is particularly desirable, but in the former it is excelled by many. The possession of extremely delicate tambours enables the observer to proceed rapidly and to obtain beautiful and accurate records which would be impossible with ordinary apparatus. The horizontally writing tambours of French manufacture are particularly delicate.

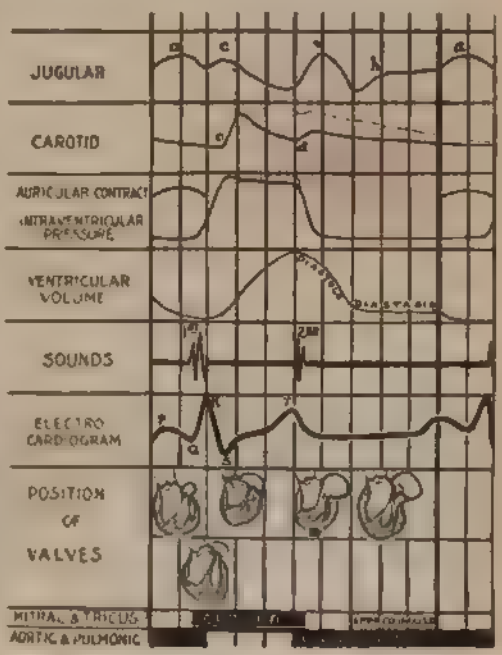

FIG. 45.—Diagram representing the various events in a cardiac cycle. Letters as in previous figures. Time in $\frac{1}{5}$ seconds, vertical lines. Dotted lines represent curves obtained when the peripheral resistance is high. The pulse becomes anacrotic and the intraventricular increases toward the end of systole.

INTERPRETATION OF WAVES UPON THE VENOUS TRACING.

The curve of venous pressure obtained clinically and in animals (Frederieq, Morrow, Hering, Theopold) corresponds exactly to those obtained within the auricles (Chauveau and Marey, Frederieq, Porter). The first

wave (*a*)[1] in the venous pulse is due to the contraction of the right auricle, and disappears when the auricle is paralyzed. It occurs about one-fifth second before the contraction of the ventricle. The onset of the ventricular contraction is marked on the venous tracing by a small wave (*c*), caused in part by the pushing up of the tricuspid valve when the intraventricular pressure rises (Hirschfelder, l. c., Bard, l. c., Morrow, Cushny and Grosh), and in part by the flow of blood from the coronary veins, which, as Porter has shown, are forcibly emptied into the auricle at this instant (Sewall and Hirschfelder,. Mackenzie thinks that it is due only to the carotid pulsation transmitted to the vein, but Morrow has obtained it after ligature of the carotid in animals. Besides the wave appears about $\frac{1}{10}$ second before the carotid wave in many cases (Hirschfelder, Bard). When the tracings are taken from the left jugular and right carotid, the *c* wave in the vein may be later than that in the artery, owing to longer time of transmission. The *c* wave is almost always present; but, as Bard has shown, it may be very small or entirely absent in hearts whose ventricles are failing (Fig. 46). The rise of the *c* wave is followed by a large fall (*x*), which may be the largest fall of pressure in the whole cardiac cycle. The exact mechanism by which this fall of pressure in the veins (and also in the auricles) is produced, and especially why it should sometimes represent the largest fall of pressure, is not clear. It is evident at this period of the cycle that several events are taking place (1) relaxation of the auricle; (2) a certain amount of downward pull which the papillary muscles

Fig. 46.—Venous tracing showing absence of the *c* wave in a case of heart failure. The tracing is otherwise normal. *J G*, right jugular vein. *C A R*, left carotid artery. Time in $\frac{1}{5}$ seconds.

Fig. 47.—A, Venous tracing showing auricular paralysis: absence of *a* wave, with large (*x*) depression during ventricular systole between *c* and *v*. B, Same tracing, faster speed.

exert upon the tricuspid and mitral valves; (3) at each systole, as can be seen when the heart is exposed, the movements of the latter within the chest are exerting a pull upon the venæ cavæ, thus pumping their contents into the auricles, (4) the outflow of blood and the decrease in size of the heart during systole cause a slight increase in the negative pressure within the

[1] Since Mackenzie's first nomenclature and lettering of the waves was introduced, a great variety of lettering and of designation by numerals has been used by different authors; but these serve to complicate rather than to simplify the question. The letters or numbers are merely symbols, and a single uniform system would be better than a Babel of terms.

thorax which may be transmitted to the thin-walled veins. It is probable that neither of these factors alone is responsible for the fall (*x* depression), but that each is active. Certain it is that auricular relaxation is not the sole cause, for as shown in Fig. 47 it may still be the largest depression in cases in which the auricle is paralyzed.

Dr Peabody has called the writer's attention to a small wave which is frequently seen during midsystole, especially in tracings from vigorous hearts, occurring just at the base of the *x* depression, and which in many cases cannot be due to lag of the lever. The origin and significance of this wave are extremely uncertain. It may be really transmitted from the artery or, as Dr Peabody suggests, may be due to slight insufficiency of the papillary muscles studied by Sewall.

The fall which leads to the *x* depression usually lasts until about the end of ventricular systole, *d* (instant of the dicrotic notch), after which it is followed by a large rise (diastolic wave of Porter; *v* or ventricular wave of Mackenzie; *vs*, ventricular stagnation (Ventrikelstauungswelle), Hering; telesystolic wave, *t*, Bard). This wave is very constant in its occurrence and is usually supposed to represent stagnation within the ventricle lasting from the end of systole until the tricuspid valve opens; the fall *v–y* indicates the opening of the tricuspid valve.

As Bard has shown, two undulations are occasionally found (*t*, *d*, *t*, telesystolic, occurring at the end of systole; and *d*, protodiastolic, occurring at the very beginning of diastole). Bard states that the wave *t* is coincident with the first secondary (predicrotic) wave of the arterial pulse, the second with the vibration of the ventricles due to the closure of the aortic valves, but this is not very satisfactory.

Sewall believes that the stagnation at the end of systole (when the upstroke of the *v* or *t* wave occurs before the end of systole) is due to a fatiguing or stretching of the papillary muscles, causing a slight tricuspid regurgitation at that instant; but in cases with no murmur in the tricuspid region this explanation needs confirmation.

The rise upon the *v* wave outlasts the end of systole by about $\frac{1}{10}$ sec., which probably represents the time required to transmit this change of pressure to the veins.

Most writers follow Mackenzie in believing that the upstroke of the *v* wave represents stasis within the ventricle lasting until the tricuspid valve opens, but cardiometer tracings show that filling of the ventricles, or at least dilatation, begins at the instant systole ends. Chauveau's tracings of the movements of the heart valves also show that the tricuspid valve opens before the time at which the crest of the *v* wave appears, so that it is probable that this wave does not represent the very instant at which the tricuspid valve opens, but that when the period *x–v* exceeds the transmission time the interval represents a period during which the venous pressure remains greater than atmospheric pressure. Or it may last until a sufficient amount of blood has entered the ventricle to have relieved the venous engorgement which followed the cessation of the factors which had produced the *x* depression.

The descending limb of the *v* wave continues as long as blood is rushing in to fill the ventricle (Henderson's period of diastolic filling), after which there is a gradual filling of the vein and a rise until the next auricular systole. In slow hearts Hirschfelder and A. G. Gibson have shown that the inflow into the auricles and the filling of the veins is no longer uniform but is interrupted by a well-defined wavelet (*h*, Hirschfelder, *b*, Gibson) which follows the *v* wave by a definite interval (Fig. 18, *h*). Both these writers indepen-

dently ascribed this wave to the snapping together of the auriculoventricular cusps at the end of ventricular filling in middiastole, and the former called attention to its correspondence

FIG. 48.—Venous tracing from a very slow heart with loud third heart sound, showing the presence of the *h* wave. *Max*, maximal blood-pressure; *Min*, minimal blood pressure.

with the onset of Henderson's period of diastasis. This fact is further borne out by the presence of a corresponding wave upon the tracing from the œsophagus (Fig. 54, *h*). This wave disappears when the pulse-rate becomes more rapid (Fig. 49).

FIG. 49.—Tracing from the same person one hour later, after giving atropine and quickening the pulse. The *h* wave is absent.

G. A. Gibson, Eyster, and the writer have occasionally seen a wave *w* in late diastole of slow pulse preceding the wave of auricular contraction (*a* wave) by a rather definite interval (Fig. 50). The distance from the *h* wave varies. This wave is assumed by the former writer to represent a contraction originating in the sinus region of the heart. Since the remnant of the embryonic sinus is actually incorporated within the body of the auricle (atrium), this view is questionable and requires experimental confirmation.

FIG. 50.—Showing a wave *w* occurring shortly before the *a* wave. (From a tracing made in collaboration with Prof. L. F. Barker.)

VISUAL EXAMINATION OF THE VENOUS PULSE.

Some of these events in the cardiac cycle may be clearly distinguished with the naked eye. Upon looking carefully at the jugular pulsation in a normal individual and placing the finger upon the carotid artery the vein will be seen to fill twice (*a* wave and *r* wave) and to collapse twice (*x* depression and *y* depression) for each beat felt in the carotid artery ("presystolic-diastolic," "physiological," "negative," "double" venous pulse (Hirschfelder)). These waves may be timed less accurately with the eye, but, although, as Mackenzie states, visual examination may save the examiner many unnecessary tracings, it should not be relied upon in doubtful cases. For example, a simple mesosystolic collapse (like that shown in Fig. 44) with absolute paralysis of the auricles may simulate a normal venous pulse.

ABNORMAL TYPES OF VENOUS PULSE.

Auricular Paralysis.—Besides this normal (negative or double venous) pulse several other types of venous pulse are seen. In venous stasis and cardiac failure the auricles may soon become weakened and the *a* wave, due to their contraction, may disappear entirely (Figs. 47 and 51). This phe-

FIG. 51.—Positive or ventricular type of venous pulse in tricuspid insufficiency, showing absence of the *a* wave. *VJD*, right jugular vein; *ACS*, left carotid artery.

nomenon is readily demonstrable in animals (v. Frey and Krehl) and need not be accompanied by any change in heart-rate, though arrhythmia is frequently present in man. In animals auricular paralysis or marked weakening of the auricular contraction may also occur as the result of vagus stimulation, so that the presence of this phenomenon alone is not always a bad omen, though usually such is the case.

FIG. 52.—Positive or ventricular type of venous pulse in tricuspid insufficiency, showing absence of the *a* wave. *JU G*, right jugular vein; *BRACH*, right brachial artery.

Positive Venous Pulse. When the auricle is paralyzed or there is a leak at the tricuspid valve, the entire form of the pulse-wave usually changes. The collapse during ventricular systole disappears and is replaced by a systolic plateau, or more usually an *M*-shaped wave with an early systolic wave *c* or *p*, a midsystolic depression, and a telesystolic wave *r* (Hewlett). Mackenzie believes that the first crest of the *M* represents a contraction of the auricle simultaneous with that of the ventricle, and that the depression in the middle corresponds to diastole of the auricle; but this form of curve has been obtained by Knoll and Theopold in animals when the auricles were stopped by vagus inhibition. This form is known as the "positive," "ventricular," or, from the fact that it appears to the eye as a single wave, the "single" type of venous pulse (see chapter on Tricuspid Insufficiency). Though the ventricular type of venous pulse occurs in tricuspid insufficiency, it is not pathognomonic of the latter and may indicate merely paralysis of the auricle.

Information furnished by the Venous Pulse.—It is apparent from the above description that the following facts are to be learned from the normal venous pulse-curve: (1) whether the auricle (atrium) is contracting, and whether each auricular (atrial) contraction is followed by a ventricular contraction; (2) the time required for the conduction of the impulse from auricle (atrium) to ventricle (the interval *a–c* on the tracing, about $\frac{1}{5}$ second in normal individuals—conduction time); (3) whether or not the tricuspid valve is closing perfectly (shown by the fall of pressure during systole and the subsequent *v* wave).[1] In irregular pulses many more important facts are to be learned from the venous pulses, which will be discussed in connection with this disturbance of function.

ŒSOPHAGEAL TRACINGS.

The venous pulse tracing reveals the conditions prevailing in the right auricle (atrium) and the state of the tricuspid valve. A corresponding investigation of the state of the left auricle (atrium) and of the mitral valve was made possible by a method used by Frederieq in animals and introduced into clinical medicine by Minkowski. Minkowski calls attention to the fact that at the level of the seventh to the ninth thoracic vertebræ (about 35 to 37 cm. from the teeth) the left auricle is in contact with the œsophagus, and when one introduces a stomach-tube to this level it receives impulses from the left auricle alone. Accordingly, an ordinary stomach-tube is capped with a thin rubber finger cot, and the latter secured by winding a silk ligature several times around it. The stomach-tube is then swallowed by the patient until it extends down 35 to 37 cm. from the teeth. It is then connected with a Marey tambour whose oscillations record the contraction of the auricle and ventricle (Figs. 53 and 54). The fall in the wave occurs when the auricle moves away from the œsophagus, the rise when it is pressed against the latter by filling with blood. Under ordinary circumstances ventricular as well as auricular systole draws the auricle away from the œsophagus so that the falls and rises correspond to auricular and ventricular systole respectively.

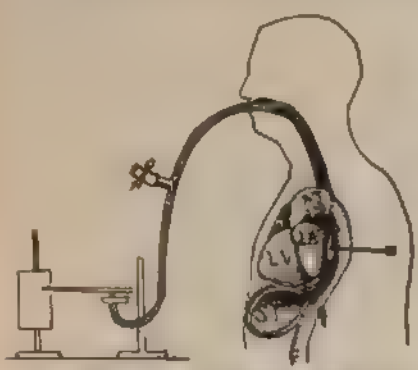

FIG. 53.—Method of taking tracing from the œsophagus to show the contractions of the left auricle. The arrow points to the thin rubber ball at the end of the œsophageal tube. ST., stomach.

Œsophageal Tracing in Mitral Insufficiency.—When the mitral valve does not close (mitral insufficiency), blood is forced back into the auricle during ventricular systole, and instead of a fall, there is a rise during systole. Minkowski's method furnishes the means for obtaining the missing link in our knowledge of the cardiac impulse and the meaning of functional murmurs, but unfortunately the swallowing of the stomach-tube is so disagreeable to the ordinary patient and so dangerous in all very severe cases as to preclude its adoption into general use. Patients can, however, often be trained to swallow the stomach-tube without difficulty, or a rubber tube of small bore may be substituted, and then very satisfactory results may be obtained.

FIG. 54.—Œsophageal and carotid tracings from a normal man.

[1] This, as has been shown by Mackenzie and by Ribl, is not absolute.

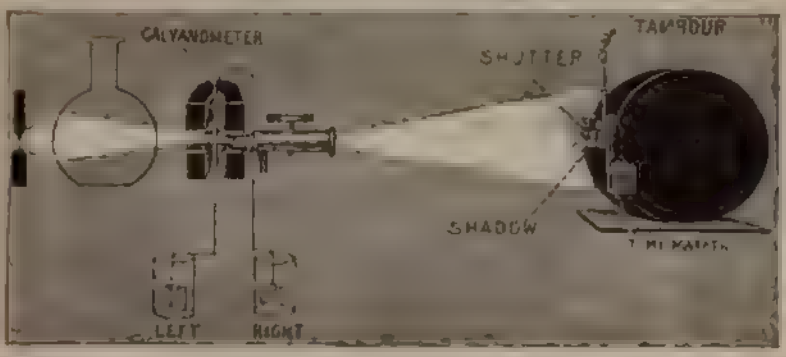

FIG. 55.—Simplest form of apparatus for recording the electrocardiogram and cardiogram simultaneously. *LEFT RIGHT*, jars filled with salt solution to receive the left and right hands respectively.

FIG. 56.—Patient with both hands placed in jars of salt solution, ready for taking electrocardiogram. (After Einthoven.)

FIG. 57.—Course of the electrical variations due to the heart-beat in man. (After Waller.) c, course of the negative wave from auricle to ventricle; b, b, zones about the auricle becoming negative during the auricular systole; a, a, zones about the ventricles becoming negative during ventricular systole.

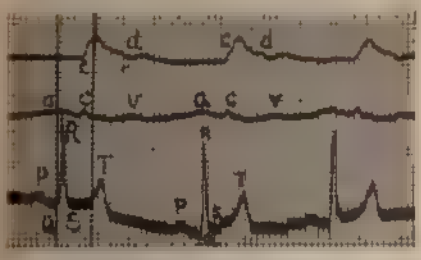

FIG. 58.—Normal electrocardiogram showing the time relations to the venous and carotid pulse-waves. (After Einthoven.)

FIG. 59.—Normal electrocardiogram tracing taken by the writer in collaboration with Prof. J. F. Barker and Dr. G. S. Bond.

INTRANASAL TRACINGS.

Mosso and also the writer have obtained very satisfactory cardiographic curves from the changes of air pressure within the thorax. These may be obtained by placing in one nostril a cork perforated by a glass tube which is connected with the recording lever. The lips are closed and the other nostril is closed by pressure. Or, the tube may be placed in the mouth and both nostrils closed by pressure. The glottis must be open and the breath held. Curves thus obtained closely resemble the œsophageal tracings in normal individuals, though the waves are smaller.

THE ELECTROCARDIOGRAM.

Another very promising method of examination which has not yet become general is the use of the electrical variations due to the heart contraction (electrocardiagram of Einthoven).

Einthoven places the patient in a chair with both hands or one hand and one foot immersed in a jar of 0·9 per cent. sodium chloride solution. Each jar is connected in the circuit with a very delicate Einthoven (or Edelmann) thread galvanometer (Fig. 55). The movements of the galvanometer are recorded photographically. At each heart contraction a series of electrical changes appear (Figs. 58 and 59), in which the first wave *P* corresponds to the auricular (atrial), the second *QR* and third *ST* to the ventricular systole. This method, at first sight the most difficult, is, when the appliances are once set up, one of the simplest of all the graphic methods. Einthoven has connected the Leyden physiological laboratory with the hospital by means of telephone wires specially laid, and is able to make his diagnoses at a distance of a mile without ever seeing the patient.

In hypertrophy of the right ventricle the wave *QR* is much larger than usual and is on the same side of the base-line as the auricular wave *P*. In cases of hypertrophy of the left ventricle the *QR* wave is inverted and its altitude is also greater than normal.

Einthoven and Kraus and Nikolai have shown that extrasystoles and other irregularities may be deciphered by this method better than by means of the venous pulse; and it is probable that it will to a great measure supplant the latter as a means of diagnosis.

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ANALYSIS OF ALTERATIONS IN CARDIAC RHYTHM.

The irregularities in rhythm of the heart may be divided first into three classes. (1) arrhythmias, in which there is no discernible order in the occurrence of beats; (2) allorrhythmias (altered rhythms), in which, though the rhythm is not regular, yet the irregular beats occur according to a certain regular system, so that the arrangement of these

beats in one section of the tracing can be prophesied from a knowledge of another, and (3) pararrhythmias (Wenckebach), in which two separate rhythms are going on in either the same chamber or in different chambers at the same time.

ALLORRHYTHMIAS.

A. Of extracardiac origin.

I. Neurogenic, due to more or less rhythmic reflex stimuli passing through the vagi and accelerators (toxic, reflex from various organs, respiratory reflexes from lungs).

a. Associated with the phases of respiration.

b. Not associated with respiration—Mackenzie's youthful type.

II. Due to disturbances in the filling and emptying of the heart from traction upon the heart and great vessels dropping of beats without heart-block, pulsus paradoxus and Riegel's pulse.

B. Of intracardiac origin.

I. Due to disturbance in the conduction of normal impulses—dropping of beats.

1. Auriculo- (atrio-) ventricular block.
2. Sino-auricular block.
3. Interventricular (?) block (hemisystole).

II. Disturbance of contractility—pulsus alternans, and failure to open the aortic valves.

III. Occurrence of beats in response to abnormal stimuli or increased irritability.

1. Extrasystoles in which irregular beat is brought on by a single abnormal stimulus.
   a. Ventricular.
   b. Auricular.
   c. Auriculo- (atrio-) ventricular
2. Permanently irregular heart.
3. Paroxysmal tachycardia (auricular fibrillation)

I. NEUROGENIC ALLORRHYTHMIAS.

Alteration in cardiac rhythm resulting from intermittent stimuli passing down the cardiac nerves constitutes one of the most common forms of cardiac allorrhythmias. As has been seen (Chapter III.), alterations of the pulse-rate may result from any stimulation of any afferent nerve, from skin, muscles, mucous membrane, and viscera, or from stimuli arising in the vagal or accelerator centres in the medulla

As Reid Hunt and Hooker have shown, the reflex stimulation may cause a slowing of the pulse-rate through stimulation of the vagus centre or under other circumstances and especially when of a different intensity, it may cause an acceleration of the pulse rate. Hunt has shown that this acceleration is due chiefly to momentary cessation of the tonic stimuli in the vagus but Hooker proves that there is also a stimulation of the accelerators. Such afferent or sensory stimuli may arise in the skin and muscles, but especially in the viscera and the serous and mucous membranes.

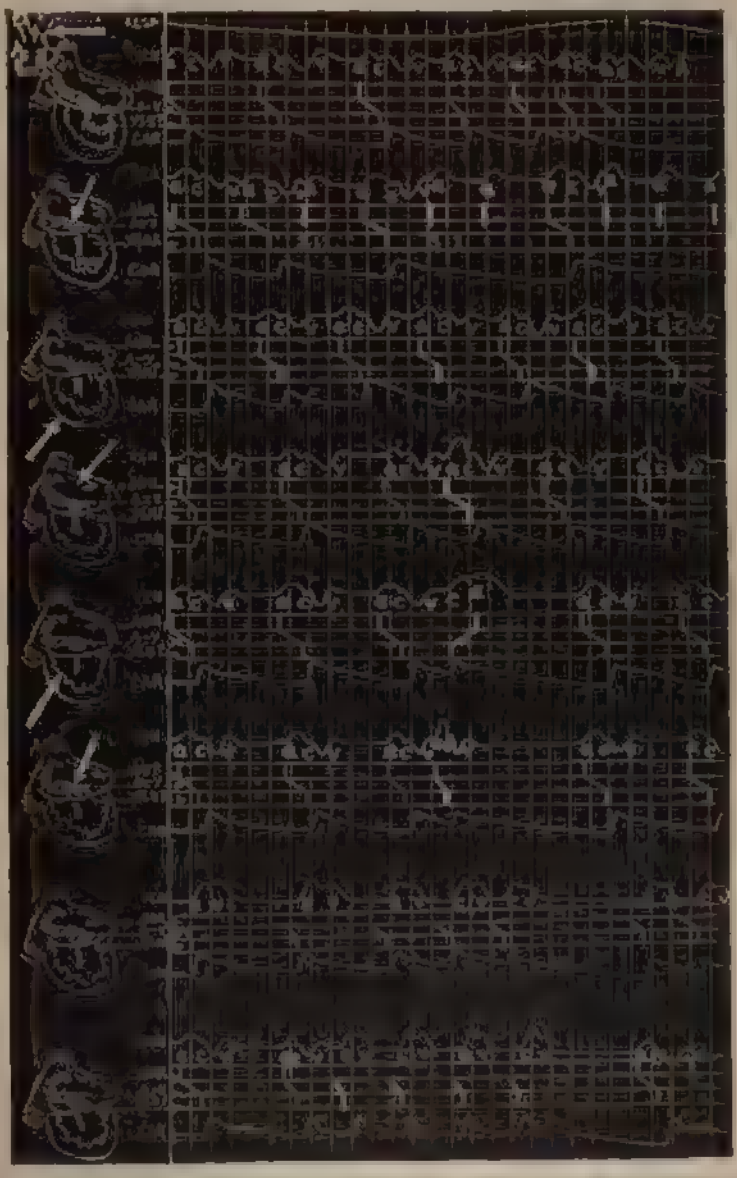

Fig. 60.—Diagram representing various types of irregular pulse. The heavy white arrows indicate the site of origin of the disturbance of rhythm. The heavy white lines indicate the course of the abnormal cardiac impulses. *RESP* respiration. *A B* auricle, *A V* or *A B* auriculo-ventricular bundle, *VENT* ventricle, *CAR* carotid pulse, *VEN*, venous pulse, *SIN* sinus region of the heart, *S V C I V C*, superior and inferior vena cavæ, respectively.

Reflex Allorrhythmias. François-Franck and Koblanck and Roesler have been able to produce such an arrhythmia by stimulating the mucous membrane of the nasal septum at a point just opposite the middle turbinate bone, and Stadler and Hirsch have done so by stimulating the walls of the stomach and intestines. There is normally a reflex slowing of the heart

during swallowing, and similar periodic slowing of the rate from stimulations of the vagus may account for many of the disturbances of rhythm in air-swallowers. Moreover, Einthoven has shown, by recording the electrical variations in the peripheral stump of the divided vagus, that, with each inspiration, afferent stimuli are passing up the vagus, and these may evoke reflex responses when the entire nervous system is abnormally sensitive.

Occurrence. — Neurogenic arrhythmias are particularly common in children and in young persons, and hence are designated by Mackenzie as the "youthful type," but this is only because the cardiac, vasomotor, and respiratory centres are in more labile equilibrium in them than in normal adults. However, whenever the nervous system becomes more irritable,—from the occurrence of visceral reflexes, emotions, or toxic influences (bacterial toxins, alcohol, tobacco, coffee, etc.),—stimuli (like those passing up the vagus) which are normally subminimal become effective. Hence allorrhythmias of this type arise in nervous individuals and in the so-called functional cardiac diseases or cardiac neuroses (Part IV, Chapter III). Since the afferent stimuli in the vagus are continually acting, it is quite natural that they should add themselves to any other afferent

Fig. 61.— Respiratory arrhythmia. (After Hewlett.)

stimuli which may also be acting, and that the alterations of rhythm will then be associated with respiration; and, since the nature of reflex responses varies with slight variations in the intensity of the stimulus, it is not surprising that there is in some cases a series of slow beats associated with inspiration and a series of rapid beats in expiration (Fig. 61), while in others the slowing occurs during expiration and the rapid beats are during inspiration. This latter type is often spoken of as normal, but in perfectly normal individuals the rate may be absolutely regular.

Reissner has shown that the irregularity is sometimes of psychic origin; or, in other words, that the stimulus exciting the cardiac nerves may descend from the cerebral cortex instead of ascending by the usual paths of afferent stimuli. This psychogenic arrhythmia is not extremely uncommon. Indeed, the writer, whose pulse has been regular at all other times, experienced such an irregularity upon one occasion of intense anxiety lasting for several minutes. The pulse became regular as soon as the anxiety passed off; and has remained so for five years, in spite of a severe tonsillitis and tonsillectomy.

As Reyfisch has shown, similar neurogenic allorrhythmias occur in meningitis and in conditions with increased intracranial tension and, as Eyster has shown, in association with Cheyne-Stokes breathing. Mackenzie has also shown that there are many other cases in which neurogenic irregularity is not associated with the phases of respiration. In

these it may be either periodic or entirely intermittent. It is most important that the exact mode of origin of such stimuli and its characteristics should be carefully studied, since this arrhythmia must be differentiated from those of myocardial origin. In these cases, though the relation of the allorrhythmia to respiration may be timed by palpation and inspection, a careful venous tracing should be made lest an extrasystolic irregularity be diagnosed when it does not actually exist.

Characteristics of Reflex Allorrhythmias. – The striking feature of these neurogenic disturbances of rhythm is that they are often characterized by instability of rhythm, by the occurrence of rhythmic changes in rate rather than by the interpolation of beats which differ from the others in character. The beats usually occur in short groups, the first beat of the slower group being the longest, the rate of the more rapid series showing a progressive increase. The last beat of the rapid series, with the vagal pause following it, may be mistaken for an extrasystole, but, on examining the few beats preceding, it will be seen that this beat was not premature and not due to an abnormal stimulus. Moreover, the beats are usually of full and almost equal strength, thereby differing from the feeble beats of extrasystoles, and they do not occur, as do the latter, abnormally early in the cardiac cycle. It is an irregularity in rhythm rather than an irregularity in force, though a certain degree of the latter may be present through the action of the vagus on the heart.

The rhythm usually becomes regular within half an hour after the hypodermic administration of atropine, .0005 to .001 Gm. ($\frac{1}{120}$ to $\frac{1}{60}$ gr.). This rule is not invariable.

When long pauses alternate with short series of rapid beats, the force of the first large beat may be slightly below that of the smaller beats, as shown by tracings with the Erlanger apparatus at or near the maximal pressure. With extrasystoles the systolic pressure of the smaller beats is usually less than that of the regular (large) beat. In both cases, however, this depends upon too many factors (time at which the extrasystole occurs, amount of systolic output, amount of peripheral resistance, factors causing the extrasystole, etc.) to be regarded as absolute criterion for diagnosis.

II. **Respiratory (Pulsus paradoxus and Riegel's Pulse).** – As will be seen in the chapters on adherent pericardium (page 506) and enteroptosis, traction upon the aorta during respiration may prevent the heart from emptying itself and thus cause the dropping of a beat in the arteries. Or, on the other hand, traction upon the great veins may produce the same effect by preventing the heart from filling. When there are adhesions in the posterior mediastinum or when the diaphragm is low, this dropping occurs during inspiration (pulsus paradoxus, Kussmaul), whereas when there are adhesions between the heart and the anterior chest wall it may occur in expiration (Riegel).

ALLORRHYTHMIAS HAVING THEIR ORIGIN WITHIN THE HEART

III. **Allorrhythmias due to Failure to Conduct Impulses generated Normally — Heart-block.**[1] — Of this there are several types. (1) Auriculo-

[1] A full discussion may be found in Part III, Chapter XI.

ventricular Heart-block.—The more usual, or at least better known, type of blocking the impulses is at the auriculoventricular junction. In this type no change occurs in the origination of the cardiac impulse or in the contraction of the auricles (atria), but the conductivity of the impulse to the ventricle by the bundle of His is impaired. Such impairment may be (*a*) functional, from overstimulation of the vagus, of which frequent

Fig. 62.—Venous tracings in heart-block. Partial heart-block (3 : 1 rhythm) during pressure on the vagus, in a case of Adams-Stokes disease.

examples are seen in every laboratory experiment. Clinically this may be seen also in the cases of digitalis poisoning and postfebrile bradycardia, especially after pneumonia and influenza, occasionally also in cases in which there is a tumor pressing upon the vagus. (*b*) Organic, from interruption of the bundle of His. In this case the block may be increased by giving atropine or anything else that quickens the heart, or it may not be affected.

Fig. 63.—Venous tracings in heart-block. Complete heart-block in a case of Adams-Stokes disease.

(*c*) There may be a combination of the two effects (v. Tabora, Gibson, Thayer), the conductivity of the injured Purkinje fibres of the bundle being still further diminished by the action of the vagus upon them, and this effect outweighing the favorable action in slowing the auricular rhythm.

The block may be partial or complete, depending upon whether the ventricles still follow the lead of the auricles or initiate their own rhythm. Thus, in the partial block the ventricles may respond to only every second, third, or fourth, or even only every sixteenth contraction, or may sometimes

respond to every second, sometimes to every fourth beat, etc. On the other hand, they may fail to contract at all over a considerable period (stoppage) during which syncope (Adams-Stokes syndrome), epileptiform seizures, or death may set in (Erlanger), or, after a stoppage of greater or less duration, they may begin to beat at a rhythm of their own, bearing no relation at all to the rhythm of the auricles (complete block). This constitutes the permanent bradycardia of Adams-Stokes disease.

(2) Sino-auricular Block.—Sino-auricular block may also occur, the cardiac impulse being generated as usual at the mouths of the great veins and coronary sinus in the region homologous with the sinus venosus of the frog, but may fail to be communicated to the auricles.

Keith and Schönberg have shown that this could scarcely be the result of a localized lesion, and would therefore depend upon the difference in the properties and irritability of auricular and venous musculature rather than organic block. The presence of such blocks is assumed by August Hoffmann in paroxysmal tachycardia, in which there is a sudden doubling or even quadrupling of the pulse-rate during the attacks, and by Hewlett in digitalis poisoning. Experimentally they have been produced by Erlanger and Blackman on the excised mammalian heart, but both Hirschfelder and Lyster and the former observers failed to do so in the heart *in situ*. Gibson assumes the existence of a similar block in a case of Adams-Stokes disease, which he cites, along with the block at the auriculoventricular junction.

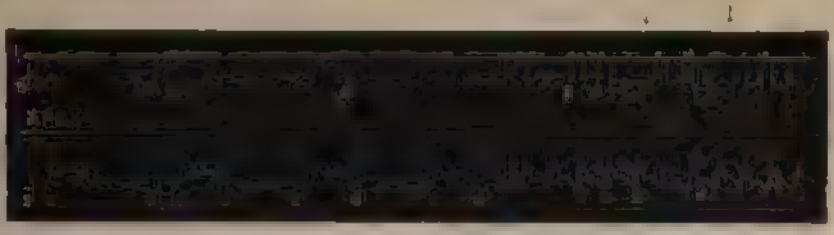

FIG. 64.—Occasional absence of apex impulse during inspiration simulating interventricular heart-block.

(3) Interventricular Block (Hemisystole).—v. Leyden in 1868 reported a case of bigeminal pulse in which he assumed that one ventricle was contracting without the other.

This case and other cases reported by the older writers, and which were undoubtedly due to extrasystoles, are really not conclusive; but recently cases have been reported by Kraus and Nikolai, and by Hewlett and Schmoll, in which the electrocardiogram and venous tracings have furnished some evidence that the right ventricle and the left ventricle may have been contracting alternately and not synchronously. For the present, one is justified in an attitude of moderate scepticism upon this point until absolute proof has been brought. Professor Barker, Dr. Bond, and the writer have repeatedly cut through the left branch of the His bundle without injuring the right branch. No asynchronism of the ventricles occurred. Extrasystoles produced in either ventricle were conducted to the other without delay. It does not, therefore, seem probable that a patch of endocarditis or myocarditis such as Aschoff and Tawara occasionally found invading a single branch of the His bundle would be able to block the impulse to one ventricle and thereby prevent its contraction. Moreover, it is possible that, like v. Leyden's, Hewlett's tracings may permit of a different and more conventional explanation.

IV. **Diminution in Contractile Power—Pulsus alternans.**—When the contractile power of the heart diminishes, or, more frequently, when the rate is increased to the point that the heart has some difficulty in carrying out effectual contractions, it is found that the alternate contractions

are of different size, some larger, some smaller, giving rise to the condition known as pulsus alternans or alternating pulse. This is especially common in the tachycardias associated with some weakness of the heart muscle,

Fig. 65.—Alternating pulse in a case of paroxysmal tachycardia.

and especially with paroxysmal tachycardia; but wherever it occurs it is an expression of disproportion between the rate and contractility of the heart (or, in Engelmann's terminology, between the chronotropic and inotropic influences).

Experimentally this can be readily shown by throwing induction shocks into the heart at a rate which it can barely follow. A pulsus alternans invariably results (Hirschfelder, Hering). After a few seconds or minutes the heart has gained its full contractility and the alternating character disappears, only to reappear when it begins to weaken. The same phenomenon is also seen in attacks of paroxysmal tachycardia (Fig. 65). Pulsus alternans is also present in some cases of angina pectoris (Mackenzie). It then indicates that the heart is in a weakened condition.

V. Dropping of Beat owing to too low Contractility. If the auricle be stimulated directly at a rate still more rapid, it can no longer follow every single stimulus, but occasionally one beat is dropped out, just as is the case in a partial heart-block, although the stimulus is being applied directly to the auricle, which intimates a little more rapidly, and it follows only alternate stimuli. If the irritability of the auricle be now suddenly increased as by pouring warm salt solution over it, it will suddenly respond with a contraction to each instead of to alternate stimuli, or it may respond occasionally to all and occasionally to only alternate stimuli, giving an alorrhythmia 1 1 + 2 1. Thus we may have alorrhythmias simulating partial heart-blocks on the one hand and extrasystoles on the other, due merely to general decrease in the irritability of the entire musculature without any special disturbance in conductivity, and just such inotrophic and bathmotrophic variations may be responsible for many of the so-called ventricular heart-blocks such as have been described by Hewlett and Wenckebach.

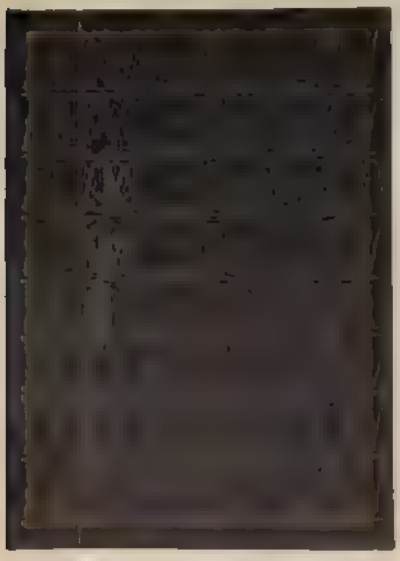

Fig. 66.—Response of frog's ventricle to abnormal stimuli. (After Marey.) Electric shock thrown into it at the instant marked by the notch in the base line and by the dotted line.

EXTRASYSTOLES.

Irregularities may be due to the origination of abnormal cardiac impulses or to abnormal response to stimuli (extrasystoles). The simplest form of this is seen in the occurrence of single abnormal beats. Experimentally it has been shown (Marey) that when a single electrical or mechanical stimulus is applied to the heart at any time except the refractory period, the latter responds

almost immediately with a contraction (extrasystole, Engelmann; premature systole, Mackenzie, Cushny, and Matthews).

Ventricular Extrasystoles.—If the extra stimulus be applied to the ventricle, the latter responds with a premature contraction, then usually but not always misses the next impulse from the auricle and pauses for a

FIG. 67.—Tracing from the jugular vein (*J. J. D.*) and brachial artery (*A. B. R.*) in man, showing ventricular extrasystoles. Time markings in $\frac{1}{5}$ seconds. *E* extrasystoles arising in the ventricle (not preceded by an *a* wave). Time of the bigeminus (regular systole + extrasystole + pause) $\frac{3.0 + 5.4}{5} = \frac{2 \cdot 4.2}{5}$.

while, until the second impulse from the auricle reaches it. We have, therefore, a normal contraction, a premature contraction, and the subsequent pause (which together may be termed a bigeminus), lasting as long as two regular contractions. The bigeminus may be spoken of as a "full bigeminus" when it lasts through two full cardiac cycles, and a "shortened bigeminus" when the duration of regular systole + extrasystole + subsequent pause is less than two cardiac cycles.

Auricular Extrasystoles.—When, however, the extra stimulus is applied to the great veins or the auricle, the bigeminus lasts less than two cardiac cycles if the stimulus follows closely upon the regular contraction, and exactly equal to two cycles if it is applied late (Hirschfelder and Eyster). If the stimulus is applied early, the auriculoventricular (atrioventricular) conduction time (*a c*) interval is slowed. Later in the cycle it is unchanged.

FIG. 68.—Tracings from the jugular vein and brachial artery of a patient with trigeminal pulse due to the regular occurrence of two auricular extrasystoles (*A. E.*) after each regular systole. The *a* wave and general form of the venous pulse are the same for the regular and the auricular extrasystoles.

The two forms of extrasystoles occur clinically and may be differentiated by the analysis of the venous pulse; the extrasystoles of auricular (atrial) origin often give rise to shortened bigemini, while ventricular extrasystoles always cause full bigemini. In the tracings of auricular extrasystoles one can see the auricular wave before the ventricular even in the extrasystole; the ventricular showing a single large wave due to ventricular systole, sometimes with the notch due to the contraction of the auricle from reversed conduction of the impulse.

Occasionally ventricular extrasystoles can be distinguished on inspection by the large flapping "single" pulsation in the jugular vein which accompanies them, in contrast to the double venous pulse of the normal beats and the auricular extrasystoles (Hirschfelder).

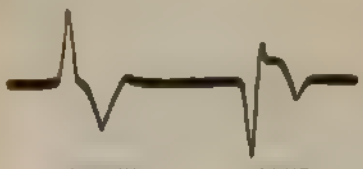

FIG 69.—Diagrammatic reproduction of the electrocardiograms obtained in the dog as the result of extrasystoles arising in the right and left ventricles. (After Kraus and Nikolai.)

A further advance in the clinical study of extrasystoles is due to the clinical use of the electrocardiogram by Einthoven and his pupils and more recently by Kraus and Nikolai, Hering, and Lewis.

Einthoven called attention to the presence of certain very peculiarly formed electrocardiograms obtained from irregularly acting hearts. Kraus and Nikolai were able to reproduce these abnormal waves by producing extrasystoles in dogs, and found that extrasystoles arising in the right and left ventricles respectively produced curves which were the inverse of one another (Fig. 69).

Kahn in Hering's laboratory has been able to confirm these findings in great part. However, he calls attention to the fact that they do not hold absolutely, and shows that stimuli applied to neighboring points in right and left ventricles, near the apex, may elicit electrocardiograms which differ only slightly from one another.

FIG. 70.—Electrocardiogram of a patient with mitral stenosis, showing extrasystoles, which arise in the right side of the heart. Taken by the writer in collaboration with Prof. L. F. Barker and Dr. G. S. Bond. Current led off from the right hand and left foot. Lettering of curves as in Fig. 9. *P R T* represent normal waves. *EXTRASYS.* extrasystoles.

Stimuli which Cause Extrasystoles.—The question as to the nature of the stimulus which gives rise to extrasystoles in man is of the greatest practical importance, for many writers (especially Fr. Muller) are of the belief that they never occur unless the heart muscle is diseased. On the other hand, Mackenzie, whose observations have been extended over a period of fifteen years, regards them as of no special significance either in prognosis or in influencing the patient's manner of life. He mentions having advised one of his patients to continue playing football in spite of his extrasystoles, and adds that the extrasystoles disappeared.

Experimentally it has been shown by Knoll, Marey, Hering, and others that ventricular extrasystoles may be produced whenever either the left ventricle or the right is prevented from emptying itself (*i. e.*, by clamping the aorta or the pulmonary artery). In man they are also most common in conditions in which there is a high blood pressure and the heart is just beginning to fail—chronic nephritis, myocarditis, aortic insufficiency, and probably fails to discharge a sufficient amount of its contents. This probably acts as a stimulus for a second extrasystole, as is frequently seen (pulsus trigeminus). Ventricular extrasystoles are most common in hearts whose rate is slow and hence which discharge a large amount of blood. They are particularly common at the end of the first third of

diastole when the filling of the ventricle is nearing completion. The ventricular fibres are stretched more or less by the influx, and in conditions of increased irritability the stretching of the fibres may act as a stimulus and give rise to the extrasystoles.

Similar conditions are observed with reference to the auricle. Dr. Cameron, in the writer's laboratory, observed an instance of permanent bigeminal pulse in a dog due to the presence of a bubble of air in the right auricle. The air had entered

FIG. 71.—Volume curve of the ventricles, showing the dilatation which followed the entrance of an air bubble into the right auricle. (Kindness of Dr. Cameron.) The extrasystoles drive very little blood into the aorta. *DIL*, dilatation.

from a hypodermic syringe during an intravenous injection. When the bubble was massaged out of the auricle the bigeminal pulse disappeared. It seems not improbable that mural thrombi may play a similar rôle, though it is certain that this is not always the case.

Auricular extrasystoles may also be produced experimentally by causing a stenosis at the auriculoventricular orifices (Hirschfelder). Clinically they occur quite commonly in mitral disease and most frequently begin at the time of the *c* wave, the very instant in the cycle at which the auricle is most distended (Fig. 68). Nevertheless, it must be confessed that much remains to be learned regarding the nature of the stimulus or stimuli, and the actual functional significance of extrasystoles.

Palpitation with Extrasystoles. — Extrasystoles are very frequently associated clinically with cardiac hyperæsthesia in the form of palpitation, so that many clinicians erroneously regard all irregularities with palpitation as extrasystolic. However, it is possible that this hypersensibility about the heart may have some causal relation, since Hornung has shown that extrasystoles in the dog are most readily produced by stimulating in the vicinity of the cardiac nerves—auriculo(atrio)ventricular and interventricular grooves—and that they cannot be produced after cocainizing the epicardium. True extrasystoles cannot be produced experimentally by stimulation of nerves (Hoffmann, Hering), and though it is occasionally claimed that they occur in gastro-intestinal diseases the exact relation is not clear. It is certain that they are often brought on by constipation and flatulence in certain persons, but whether there is a myocardial lesion already present in these cases is a still open question.

Diagnosis of Extrasystoles. In some cases it is very difficult to distinguish between the neurogenic irregularities and the auricular extrasystoles. Dehio has called attention to the fact that the former disappear under the administration of atropine, while the latter remain unaltered. Nevertheless one cannot always be certain that the dose of atropine, even if it has given rise to symptoms, has been large enough to produce the effect.

An example of this type was present in a patient seen by the writer several years ago, in whom palpitation and arrhythmia had been present for several years, the patient being conscious not only of the occurrence but also of the size of every beat, and noticing especially a group of one large beat with two small ones followed by a pause (pulsus trigeminus). The tracing made while the patient was in the hospital showed that these were due to a single beat followed by two smaller and earlier ones and then by a pause, all the beats being preceded by an auricular contraction (*a* wave). This irregularity was much more marked whenever the patient was constipated, but it also persisted after 0.5 mg. ($\frac{1}{120}$ gr.) atropine, which gave the patient marked symptoms but caused no change in rate. In such a case it is very difficult to state whether we have to deal with auricular extrasystoles or with a very rapid pulse interrupted by variations in diastole (youthful type). The ineffectiveness of atropine and the extreme irregularity are against the latter view. However, the diagnosis of extrasystoles can usually be made by means of the electrocardiogram.

In the routine physical examination extrasystoles may often be diagnosed on auscultation by the abrupt change from a rhythm 1 - 2 - 1 2 1 2, etc., to 1 2 3 4 1 2, etc. Accompanying this there may be seen an early wave in the jugular pulse. In the case of auricular extrasystoles the pulsation does not differ from the double pulse of the regular beat, but with ventricular extrasystoles there is a single wave, large, conspicuous, and flapping.

Ineffectual Contractions.—When the extrasystole occurs early in diastole, the heart may not have recovered from the effect of the last systole sufficiently to generate a forcible contraction. The aortic valves are not opened. The aortic second of the extrasystole disappears and the sounds change from 1 - 2 - 3 4 1 2, etc., to 1 2 3 - - 1 - 2, etc. By beating time to the regular beats it is sometimes possible to note that the total rhythm is unchanged by occasional ventricular extrasystoles. Such extrasystoles correspond to impulses on the apex and jugular tracings but not on the carotid.

The variations in the force of the extrasystoles or in the beats of the absolutely irregular pulse are great. Occasionally, especially when the extrasystoles occur early in the cardiac cycle and there is a high peripheral resistance, the intracardiac pressure may not reach the aortic pressure and the aortic valves are not opened. The systole has been ineffectual (Frustrane contractionen, Hochaus and Quincke). The compensatory pause after these may be so long and the circulation may be so poor that actual syncope simulating the Adams-Stokes syndrome (W. B. James) may take place in the interval between the regular beats. On the other hand, a great deal of cardiac energy has been expended without opening the cardiac valves and without propelling any blood. This increases the cardiac fatigue.

Bigeminal and Trigeminal Pulses due to Extrasystoles. Very common forms of extrasystolic irregularity are those in which the extrasystoles occur after each regular beat; thus we may find every beat followed by a single extrasystole and compensatory pause, so that the pulse beats occur in pairs separated by pauses (pulsus bigeminus), or there may be two extrasystoles following regularly after each regular systole (pulsus trigeminus), as in Fig. 68. These may be of either the auricular or the ventricular type, dependent upon the site of the origin of the irregularity or of the so-called auriculo(atrio)ventricular type referred to below. As stated above, it is sometimes difficult to differentiate the auricular extrasystolic

groups from the youthful type of arrhythmia, but this may usually be accomplished by the use of a sufficiently large dose of atropine.

As Hering has shown, ventricular extrasystoles frequently disappear under atropine or any other influence by which the pulse-rate is accelerated,

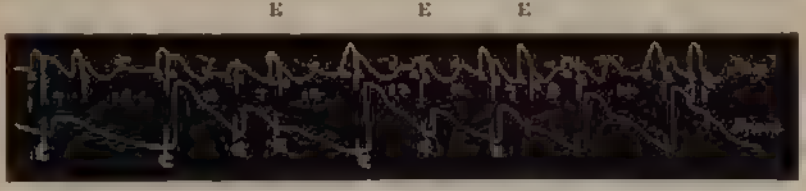

Fig. 72.—Extrasystoles with shortened conduction time, supposed to arise in the auriculoventricular bundle.

so that the normal stimuli fall in at about the periods at which the abnormal stimuli would have fallen. The form of the venous pulse in ventricular extrasystoles is, however, characteristic.

Fig. 73.—Variations in conduction time (a–c) in a case of mitral stenosis.

Auriculo(Atrio)ventricular Extrasystoles. It is also claimed by Hering and Rihl, Mackenzie and Wenckebach, Lohmann, Schmoll, Mackenzie and Morrow, and others that extrasystoles may arise in the Purkinje cells of the conduction system, and that such extrasystoles are characterized by a shortening in the conduction time (a–c interval on the venous pulse). Extrasystoles with shortened conduction time are not extremely rare, and it is possible that this explanation may be correct, but it is not founded upon any direct experimental proof.

Hering, who originated the doctrine, observed such extrasystoles occurring spontaneously in apes, but did not clear it up by any experiments. Gaskell has shown in frogs that if the tissue at the auriculoventricular junction was touched with a probe a series of extrasystoles set in in both auricles and ventricle. Lohmann also observed them persisting after the tissue in the vicinity of the His bundle had been stimulated. In a later investigation upon the excised heart Lohmann poisoned the region of the vena cava by means of cotton soaked in formalin. He then sometimes saw extrasystoles set in spontaneously. The auricles and ventricles sometimes contracted simultaneously, sometimes there were ventricular extrasystoles.

Hirschfelder has repeatedly produced extrasystoles with shortened conduction time by faradic stimulation of the auricular appendix. The appearance of such extrasystoles after faradization, in excitable hearts, in the intervals between paroxysms of tachycardia in man, etc., seems to correspond with a state of greatly heightened excitability. Whether the actual stimuli arise in the cells of the sinus region or in those of the conduction system is still uncertain. It is not improbable that there may be an increased irritability of all the primitive cardiac tissue (sinus and conduction system), and that in the sinus this manifests itself by the generation of abnormal stimuli, while in the conduction system it is shown by increased speed of conduction.

Upon the clinical side there is little positive evidence. Peculiar extrasystoles often occur between attacks of paroxysmal tachycardia, but occasionally also in cases with simple valvular lesions. Keith has found patches

of fibrous myocarditis in the vicinity of the His bundle in cases which had shown these extrasystoles, and thinks that they irritated the cells in the vicinity, but such scars are very common, and elsewhere in the heart are not known to act as irritative lesions. Moreover, the writer has never been

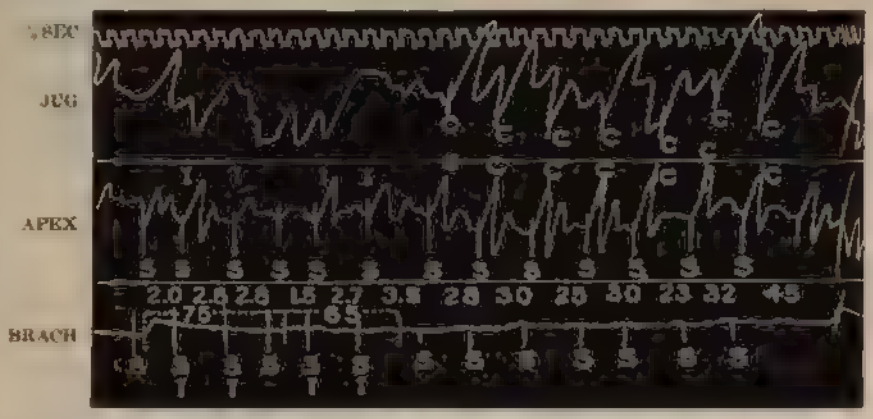

FIG. 74.—Tracing showing absolute irregular with weak ineffectual systoles (*I, I, I*) which do not open the aortic valves. *S* onset of ventricular systole. Numerals refer to duration of cardiac cycle in tenths of a second. The venous pulse is of the ventricular type.

able to produce them by pressure upon the bundle with an Erlanger clamp, injection of mercury into the left branch of the bundle, etc. So that in spite of the interest in the subject it must be admitted that the occurrence of extrasystoles with shortened conduction time cannot be as yet regarded as absolute proof of a lesion near the His bundle.

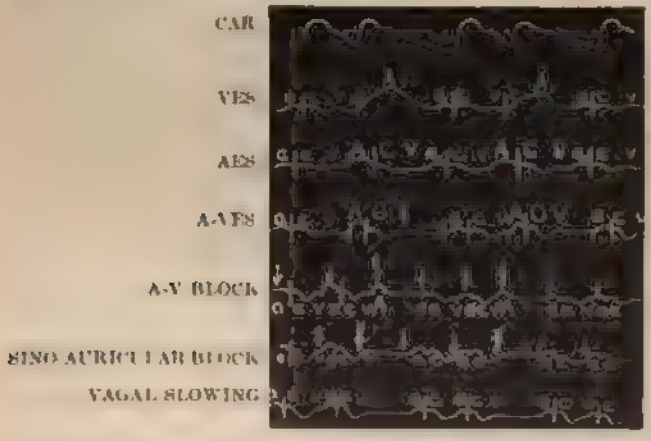

FIG. 75. Diagram showing the alterations of rhythm which may cause a pulsus bigeminus. The arrows indicate the incidence of stimuli on

Various Types of Allorrhythmia which may Result in a Bigeminal Pulse.—It must be borne in mind that the bigeminal pulse is not pathognomonic of any single disturbance of function, but may occur in any of the following conditions (Fig. 75): (1) recurring ventricular extrasystoles; (2) recurring

auricular extrasystoles; (3) recurring auriculoventricular extrasystoles; (4) recurring slight auriculoventricular heart-block, the ventricle failing to follow every third beat; (5) recurring sino-auricular block (?), the auricles failing to respond to every third impulse; (6) recurring vagal prolongation of every alternate diastole. Similar conditions hold for the trigeminal pulse except that two extrasystoles or regular beats are interpolated before the pause.

It is evident that these conditions must be carefully differentiated from one another by means of the venous tracing or electrocardiogram and atropine tests before attempts to remedy them should be begun.

PARARRHYTHMIAS.

In some of the allorrhythmias separate rhythms may be noticed in the different chambers, either conducted to one another and interfering periodically, or not conducted (heart-block). Wenckebach, who first called attention to this, has proposed the name p a r a r r h y t h m i a for these forms. The simplest example of this would be the bigemini. Another example would be seen if, without loss of conductivity, spontaneous contractions would occur in the ventricles at the usual slow rate, and these go on simultaneously with the regular beats following the auricles, though with occasional pauses due to interference. Cushny has shown this to occur in digitalis poisoning, and it is not improbable that it may explain many otherwise undecipherable arrhythmias, though little work has been done along these lines up to the present.

ABSOLUTE ARRHYTHMIA.[1]

Next to the neurogenic allorrhythmias the most common form of irregular heart action is the permanent irregularity (disorderly rhythm, Mackenzie; pulsus irregularis perpetuus, Hering; arrhythmia perpetua, Gerhardt)

Fig. 76.—Absolute permanent irregularity with *a* wave preserved in a case of mitral stenosis.

This represents, as Mackenzie has shown, the common type of chronic arrhythmia seen in old cases of myocarditis and of valvular lesions. As the result of chronic stasis there is a permanently high venous pressure which brings about dilatation and paralysis of the auricles. The *a* wave is absent from the venous tracing (Mackenzie) (Fig. 76), from the œsophageal tracing (Hewlett), and the corresponding wave has disappeared from the electrocardiogram (Hering). There is probably a perpetual or a transitory paralysis of the auricles (atria). As Hering's electrocardiograms show, the arrhyth-

[1] The term perpetual irregularity is inaccurate, since it is sometimes transitory.

mia is due partly to extrasystoles, which are shown by their characteristic curves, and partly to periodic (respiratory) alterations in the regular beats.

The site at which the cardiac impulse originates in this irregularity is a matter of some dispute. Mackenzie believes, without further proof, that the site of automaticity is shifted from the sinus region of the auricle (atrium) to the cells of the His bundle ("nodal rhythm"), but it has not been shown that, just because the auricular contraction and the corresponding negative wave are absent, the cardiac impulse is not arising in the region of the sinus. According to Mackenzie, the auricle and ventricle are beating simultaneously in such cases. Cushny (Heart, vol. i) has shown that such simultaneous contractions actually occur in experimental aconite poisoning. On the other hand, v. Frey has shown that the auricles become paralyzed at about 20 mm. Hg pressure, and observations by Dr. Hooker, as well as by Mr. C. C. Cody, indicate that in cases with permanent arrhythmia the venous pressure often approaches this level.

FIG. 77.—Perpetually irregular pulse with absence of *a* wave.

Radasewsky, under Dehio's direction, was the first to call attention to the occurrence of fibrous changes in the auricles under these conditions, but the exact relations were shown by Schönberg, who studied careful serial sections of the entire veno-auricular region in five cases that had been studied clinically by Gerhardt during life. Schönberg confirms Radasewsky and finds definite patches of infiltration about the veno-auricular border. On the other hand, G. Müller has reported a case in which the entire musculature of the auricles had disappeared and the rhythm had remained regular.

Clinically, one sometimes sees an acute onset of absolute arrhythmia with paralysis of the auricles, especially in the acute heart failure of mitral stenosis. This state may last only for a day or so and disappear under treatment; on the other hand, it may last for weeks, perhaps for months, and then disappear. The longer it persists the greater is the probability of serious changes and the less that of recovery. On the other hand, the presence of an absolute and apparently permanent arrhythmia with auricular paralysis is perfectly compatible with a fair degree of vigor.

This is shown by a medical student now under the writer's care, who has had an irregular pulse of this type for two years, during which only occasional auricular waves have been obtainable upon his venous pulse, and these during his periods of greatest vigor. He has had slight shortness of breath on exertion, but no enlargement of the heart, murmurs, or other signs of organic heart disease. His arrhythmia did not disappear under .00075 Gm. (1/80 gr.) atropine subcutaneously. The electrocardiogram does not reveal any extrasystoles. His trouble does not date from any acute infectious disease, nor from any overstrain other than the long hours of work in a hospital. The nature of the lesion and its significance in cases like this are still mysterious, but it is possible that in such cases there may be a patch of myocarditis in the sinus region of the auricle like those shown by Schönberg.

Permanent arrhythmia with persistence of the auricular contraction (a wave upon the venous pulse) is met with occasionally in mitral disease, especially in mitral stenosis; and represents one form of the so-called

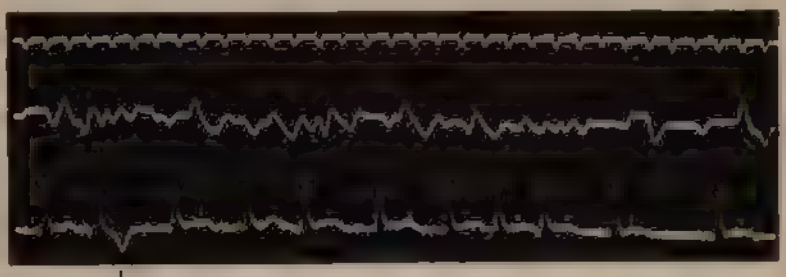

FIG. 78.—Electrocardiogram from a case of perpetual absolute arrhythmia, showing extrasystole (After Hering, *Deutsch Arch. f. klin. Med.*, xcv.) *E* extrasystole, *R T* electrical waves. The *P* (auricular wave) is absent.

"mitralized pulse." In such cases it may at first seem absolutely impossible to analyze the arrhythmia, but from time to time definite groups of systoles may be discerned. Sometimes these groups are produced by the occurrence of varying numbers of auricular extrasystoles, sometimes by the occurrence of a few beats in which there is doubling of the pulse-rate and shortening of the conduction time, just as is seen in paroxysmal tachycardia. Since Hering has demonstrated the extrasystolic origin of the absolute arrhythmia with auricular paralysis (Mackenzie's "nodal rhythm"), and since Hirschfelder has produced the latter in experimental mitral stenosis by bringing about extreme stasis in the left auricle, it seems not impossible that the question of auricular paralysis may be one of the degree rather than of the character of the disturbance.

The prognostic importance of a permanent arrhythmia with auricular paralysis depends, like all other cardiac conditions, chiefly upon its effect upon cardiac function.

MECHANICAL EFFECT OF ARRHYTHMIA IN THE CIRCULATION.

The mechanical effect of any arrhythmia is to slow the circulation, as may be easily seen from the volume curve of the heart during a period of irregularity. This slowing in itself tends to bring on cyanosis, increase the CO_2 in the blood, and, as Cameron has shown, to diminish the tone of the heart muscle in this way. On the other hand, the long pauses cause an increase of pressure in the veins, and the influx of venous blood under a relatively increased pressure acting upon cardiac muscle, whose tone is diminished, tends to overdistend the heart (as seen in Fig. 79). The overdistention, by increasing the diameter

FIG. 79.—Effect of arrhythmia on the circulation: blood-pressure and volume of the ventricles. Tracing from a dog's heart stimulated irregularly with induction shocks. *I I I*, ineffectual systoles. *DIL* dilatation. Upstrokes on volume curve represent outflow from the ventricles.

of the ventricular chamber, increases the hydrostatic pressure upon its walls and causes it to work at a disadvantage. Thus is established the vicious circle of the irregular heart:

Overfilling of heart
↑ ↓
Slowing of circulation — Irregularity

The effect is most marked in the auricles, where tone changes show themselves in more marked degree than in the ventricles, and the diminution in their tonicity hastens their paralysis. When the auricles are paralyzed, the genesis of efficient stimuli becomes more difficult, it is harder to accelerate the pulse during exercise, etc., and consequently it becomes easier for CO_2 to accumulate in the blood in the irregular than in the regular heart, and the heart in this condition is *per se* permanently weakened.

Effect of Digitalis in Absolute Arrhythmia.—The good effect of digitalis in this condition lies not in affecting the rhythmicity but particularly in restoring tone and force of the heart-beat, thus reversing the vicious circle. The pulse becomes more regular, sometimes entirely regular. When the rhythmicity is destroyed by permanent paralysis of the auricles it never returns, but the general cardiac condition may be benefited by increase in tone and strength. On the other hand, when the muscle-fibres are in too bad condition, they are oversensitive to digitalis and a small dose causes them to pass not into the first but into the second or third stage of digitalis poisoning.

PAROXYSMAL TACHYCARDIA.

Another group of allorrhythmias which may be classed with the extrasystoles is that in which there is more or less paroxysmal increase in pulse-rate, frequently amounting to exact doubling of the rate, suddenly taking place and suddenly subsiding.

This condition is seen in paroxysmal tachycardia (Hoffmann) and in paroxysmal irregularity (Cushny and Edmunds), and in the latter condition has been shown to be associated with fibrillation of the auricles. Experimentally it can be brought on by stimulating the auricles with a strong faradic current, the auricles then going into very rapid more or less fibrillary contractions, the ventricles following at a fairly regular rate which is almost exactly double the previous rate, this rhythm persisting for several minutes after the faradization has been stopped and then suddenly halving. During its continuance it may or may not be stopped by maximal stimulation of the vagus, just as is the case clinically in paroxysmal tachycardia, but seems to yield at once to strophanthus intravenously. The nature of this sudden doubling is peculiar. It also occurs in the ventricle upon faradizing the ventricular muscle directly, and similar exact doubling and exact halving of rate have been observed in the frog by Engelmann and in the mammal by Trendelenberg. (See Part IV, Chapter I.)

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V.

X-RAY EXAMINATION.

The discovery of the X-rays by Röntgen in 1895 introduced a new era in cardiac diagnosis. By this means we can now actually see the heart, observe its outlines with accuracy, and note the changes of position and of contour with different phases of respiration, and even to a certain extent the changes from systole to diastole. All these data, when obtained with proper precautions, are absolutely accurate, and have greatly supplemented the observations made by percussion.[1]

METHODS OF EXAMINATION.

Most of the facts desired in the study of the circulatory system with the X-ray may be gained by means of inspection with the fluoroscope, a screen of barium platinocyanide or calcium tungstate which is rendered luminous wherever the X-rays strike it.

A tube of low vacuum ("soft tube") should be used, one which shows the bones of the hand black without revealing their internal structure, and the tissues of the hand a fairly dark gray. The patient's chest wall should be at least 50 cm. from the screen. Recently the usual distance has been increased to 2 M. (6½ ft.), at which the rays are almost parallel.

It is sometimes best to interpose a lead screen, with adjustable opening, between the patient and the tube in order to cut off all the rays except those emanating from a small part of the anticathode, thereby securing the greatest possible definition of focus. Indeed, Immelmann found greatest definition when the opening in the lead screen was only 1 cm. Often a lead cylinder (Albers-Schoenberg) is very satisfactory. It is also important that no large objects be placed near the cathode, as rays striking these may also generate secondary rays which affect the fluoroscope or photographic plate and thus blur the outline of the original image (Walter). For securing sharp images it is preferable to keep a number of tubes with vacua of different degrees which may be interchanged, rather than change the vacuum in each tube. Changing the latter shortens the life of the tube by heating the platinum target and causing the latter ultimately to become bent, so that the rays are not reflected uniformly from its surface.

The X-ray image is a true shadow formed by the cutting off of rays and not by their refraction, and the shadow is magnified in proportion as the object is nearer to the tube or farther from the fluoroscopic screen.

[1] Examinations with the X-ray require a very special technic, for which the student is advised to consult the special text-books upon the subject, especially:

Albers-Schoenberg. Die Rontgentechnik. Hamburg, 1906.

Gocht, H. Handbuch der Rontgenlehre zum Gebrauche fur Mediziner. 2te Aufl., Stuttgart, 1903.

Williams, F. H. The Röntgen Rays in Medicine and Surgery. New York, 1903.

Beck, C. Rontgen ray Diagnosis and Therapy.

Kassabian. Rontgen Rays and Electro-Therapeutics. Phila., 1909.

In fluoroscopic examination it is most important for the observer to accustom his eyes to the darkness before turning on the current. It is a well-proved physiological fact that the longer the sojourn in darkness the greater the delicacy of vision. Hence the examiner's vision is improved by closing or blindfolding his eyes or by going into a dark room some ten or fifteen minutes before the patient, and the examining room should be lighted only enough to permit the patient to undress and assume the proper position. The examiner may also keep his head under a dark hood or wear heavily smoked glasses during this time so as to accustom his eyes to the darkness.

In looking over the areas of light and shadows each region should be studied carefully and in detail, the size and shape of the shadow, the clearness of the outline, and the distribution of areas of half shadows as well as of full shadows. Not of least importance are the so-called pulmonary figures, the half-tone shadows of pulmonary vessels, of bronchial glands, and of strands of adhesions.[1] Not only the full shadows but especially these half shadows should be examined with care, for an interpretation not apparent at first may become clear after a few minutes' observation.

Radiographers are, moreover, in the habit of looking at the fluoroscope through half-closed eyes in order to intensify the contrast. This may be further intensified by the use of dark glasses. The writer has also found it very useful to look at the shadow or skiagraph through a biconcave lens which at once sharpens the contours and intensifies the contrasts.

Often an area may be indefinite during quiet breathing or expiration and become quite definite on forced deep inspiration, or it may become so by simply turning the patient so that the rays pass through his body in a different direction. These and similar precautions, like a careful physical examination, reveal the unsuspected, and distinguish the skilled examiner from the unskilled.

Fig. 80.—Radiograph of normal chest. After v. Ziemssen and Rieder. (To be held at the chest plate in front.

The Cardiac Shadow.—The heart shadow thrown upon a screen at the front of the chest is shown in Fig. 81. It will be noted that the outline of the shadow closely resembles the area of relative dulness on percussion, except that the former extends upward over the manubrium sterni, where it is due to the presence of the latter and of the great vessels and not of the heart. In the second left interspace the shadow of the pulmonary artery is seen, and in the second right that of the aorta. Occasionally a small prominence is seen to the left of the sternum arising at the arch of the aorta. This is sometimes mistaken for an aneurism, but if the patient be turned a little it will be seen to be due to the curving of the aorta (Holzknecht).

Oblique Illuminations.—Much can be learned by turning the patient about and examining him in several planes, as was first performed by v. Criegern and Holzknecht (l. c.), and later by Rieder, who suggested the

[1] See chapter on Adherent Pericardium.

following cardinal directions (Fig. 81): (1) dorsoventral; (2) ventrodorsal; (3) sagittal from right; (4) sagittal from left; (5) from right posterior to left anterior; (6) from left posterior to right anterior; (7) from left anterior to right posterior; (8) from right anterior to left posterior.

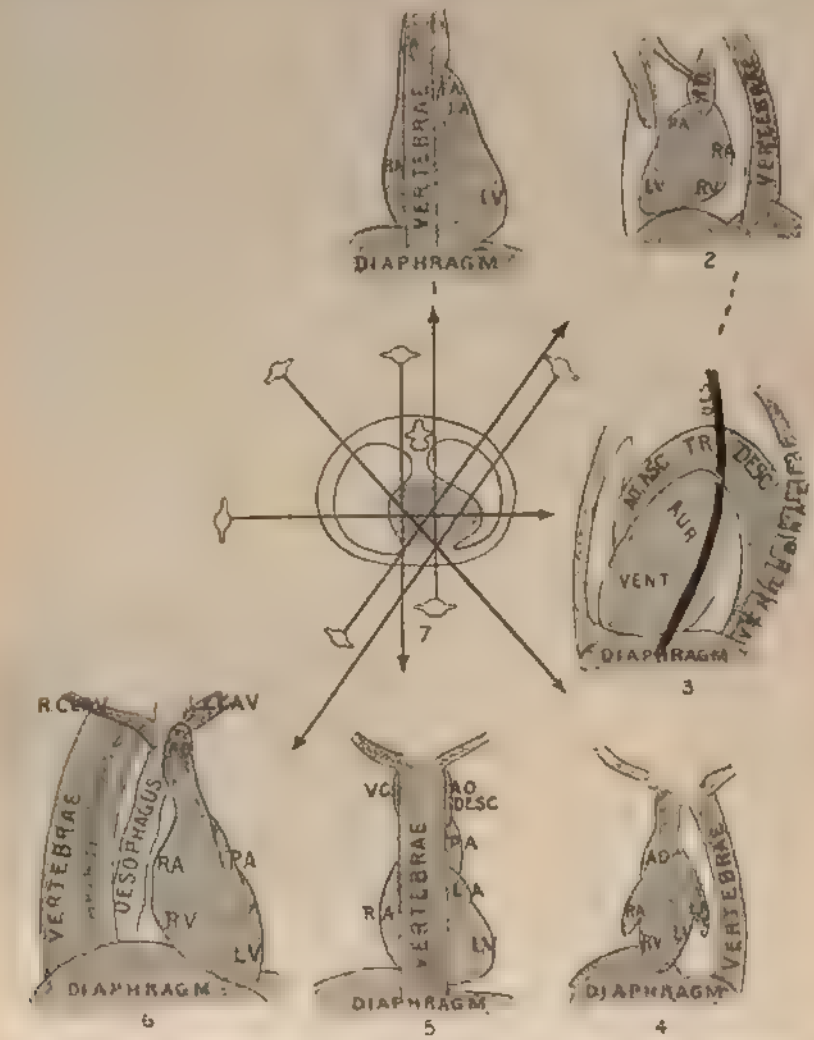

FIG. 81.—X-ray shadows in different axes of the body. (Modified from Holzknecht.) The arrows show the direction of illumination and position of the tube corresponding to the shadow. *AO*, aorta; *PA*, pulmonary artery; *LA*, left auricle; *RA*, right auricle; *LV*, left ventricle; *RV*, right ventricle. In 3 there is a metal sound in the œsophagus.

By the examination in these planes every part of the heart can be brought into view, even the left auricle, which escapes observation in almost all other methods of examination but appears quite clearly when the tube is placed at the back or behind the right scapula. The oblique and transverse examinations should never be omitted.

THE ORTHODIAGRAPH.

The shadow of the heart and vessels upon the screen or plate is always larger than the objects themselves. In order to obviate this when measuring out the heart F. Moritz devised an instrument known as the orthodiagraph (Fig. 82).

Fig. 82.—A simple form of orthodiagraph. (After Gillet.)

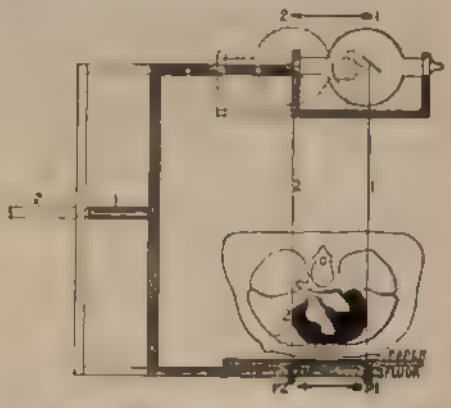

Fig. 83.—Diagram showing the use of the orthodiagraph. 1 first position 2 second position. *P*, pencil, *FLUOR*, fluorescent screen.

In the orthodiagraph the fluorescent screen and X-ray tube are fixed upon each arm of a large *U*-shaped frame in such a way that the patient may stand or lie between the two arms of the *U* and the rays thus pass through his body to the screen. At the point upon the screen which is exactly opposite to the centre of the anticathode or target of the tube, a small hole is pierced, and a skin pencil is fixed in place here so that the site of this spot can be marked upon the body of the patient. The whole *U*-shaped frame bearing the tube and fluoroscope is movable in two directions by any one of a variety of mechanisms, so that the perpendicular ray can be brought opposite any desired point. A series of points corresponding to the exact contour can thus be marked off, and when these are joined with lines the exact outline of the heart is represented. As shown by Fig. 83, this furnishes a means of determining the size of the heart or any organ with absolute accuracy.

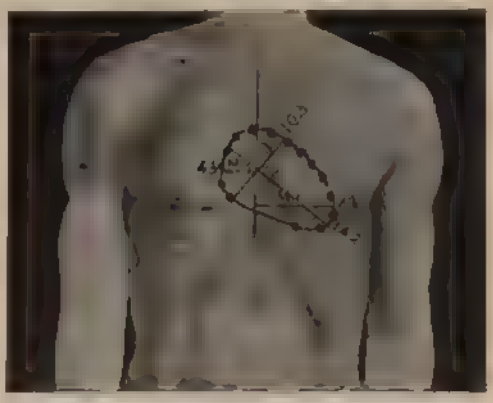

Fig. 84.—Orthodiagraphic outline of normal heart, showing Moritz's conjugates. *MR*, median to right border greatest distance. *ML*, median to left border, *L* oblique longitudinal *Q*, transverse, numerals indicate centimetres.

The outlines and mobility of the heart thus obtained are discussed on page 97.

It is also possible with the fluoroscope to watch the individual contractions of the heart and to note the changes in size due to systole and

diastole, but this is very difficult and can rarely be done with satisfactory accuracy. On the other hand, the contractions of the auricles can be seen with considerable definiteness, and dissociation of rhythm, heart-block, can often be diagnosed in this way by simple inspection (Kraus, Gibson).

PERMANENT RADIOGRAPHS.

For obtaining permanent photographs a "medium soft" tube (Moritz scale W 6 B W 5) is used in connection with a Wehnelt electrolytic interrupter and an induction coil with proper self-induction yielding a 40 to 60 cm. spark. The patient is laid upon a table with the tube above or below him, as is most suitable to the purpose of the examination. In order to absolutely immobilize him it is well to support the shoulders upon sand bags. He may also be examined standing by immobilizing the shoulders to prevent blurring of the picture. Magnification of the shadow may be obviated by placing the tube at a distance of 2 M. Skiagraphs of the chest made with very short exposures have proved particularly valuable, since they give greater definition (Rieder).

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v. Criegern. Ergebnisse der Untersuchung des menschlichen Herzens mittelst fluorescierenden Schirmes, Verhandl. d. Kong. f. innere Med., Wiesbaden, 1899, xvi, 302.

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Levy-Dorn. Schutzmassregeln gegen Röntgenstrahlen und ihre Dosirung, Deutsche med. Wchnschr., Berl. und Leipz., 1903, xxix, 921.

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Gillet. Ein Orthoröntgenograph einfacher Konstruction, Fortschr. a. d. Geb. d. Röntgenstrahlen, Hamb., 1906, x, 114.

For the examination of the heart cf. also

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VI.

PHYSICAL EXAMINATION.

While it is impossible to enter into a treatise upon physical examination, a few points which are of special importance in cardiac cases may be discussed.

General Appearance.—The general appearance of the patient, expression and color, are of great importance. The position which he naturally assumes in bed, the presence or absence of dyspnœa and orthopnœa, the general degree of nervousness or dulness are all to be noted.

The typical appearance of the cardiac patient (*cardiac facies*) is characterized by an anxious expression, bright eyes with moist, glistening conjunctivæ, cheeks full rather than sunken as in the abdominal facies, and as a rule a tinge of cyanosis about the lips.

There are two main types: (1) the *mitral* (or *mitrotricuspid*) *facies*, with rosy, flushed cheeks, dilated capillaries, and cyanosis (most commonly seen in mitral stenosis); and (2) the *aortic facies*, with pale, often sallow, rather sunken cheeks, bright eyes, moist conjunctivæ, and slight cyanosis of lips and fingers. To these might be added (3) the subicteric facies of broken compensation, with pallor, subicteric conjunctivæ, and cyanosis of the lips.

Nasopharynx.—The tonsils and posterior nasopharynx should always be carefully examined. The former are the chief portals of entry for the germs of rheumatism, while *adenoids* and *affections of the nasal septum* may of themselves induce cardiac arrhythmia, and may also be an important contributing factor in the attacks of asthma in organic heart disease.

Ophthalmoscopic Examination.—The eye-grounds should always be examined when arteriosclerosis (page 260) or congenital heart disease (page 438) is suspected.

Neck.—In the neck especial attention should be directed to the visible throbbing of the carotids, the fulness of the neck, and the size and consistency of the thyroid gland (page 585), the presence of thrills and murmurs over vessels or thyroid, or a tracheal tug (page 533). The jugular pulsation is discussed in full in Part I, Chapter IV, page 49.

Chest.—The form of the chest is of considerable importance, not only as regards kyphosis, but particularly as to its *fulness* or *flatness* (see Part III, Chapter III). In recording this, the width of the costal angle should be noted, but the *general obliquity of the ribs* in quiet expiration should be designated by noting the *vertebral spines which are on the same level with the sternoxiphoid articulation* (normally at the level of the eighth thoracic spine) (page 598). It should be noted whether the chest in quiet breathing approaches more nearly to the position of expiration, flat chest, or to that of inspiration. Pulsations, bulgings, heaving, or retractions of the ribs or interspaces, as well as the presence of abnormal shocks and thrills, should of course be noted.

Abdomen.—In the abdomen the important features to be noted are presence or absence of ascites, *enlargement of liver* (systemic

stasis), pulsation of the liver, systolic impulse (tricuspid insufficiency), systolic retraction (dilated or hypertrophied right ventricle), the nature and the time of epigastric pulsation (systolic elevation being transmitted from the abdominal aorta, systolic retraction indicating dilated hypertrophied right ventricle). A palpable spleen of cardiac origin points to infarction, septic or thrombotic. When aneurism or arteriosclerosis is suspected the course of the abdominal aorta should be mapped out by deep palpation with both hands, one above each side of the aorta and that vessel between them (page 550).

The genitalia should of course always be examined for signs of gonorrhœa and lues,—urethral smears for the former and a Wassermann reaction for the latter being made whenever possible.

Extremities.—Upon the extremities the presence of œdema and arthritis, acrocyanosis or pallor, and the size, consistency, and uniformity of the brachial, radial, femoral, popliteal, and dorsalis pedis arteries are the chief points of importance.

THE CARDIAC IMPULSE.

Mechanics of the Cardiac Impulse.—The apex itself, as shown by Ludwig and Dogiel, does not move appreciably up or down during systole; and, as Hesse has demonstrated, the transverse diameter of the heart shortens more than the longitudinal.

The chief movements which lead to the production of the apex impulse are due more to the systolic erection of the heart upon the great vessels than to its diminution in size.

Fig. 85.—Movements of the heart leading to the protrusions and retraction during systole. Forces shown by the arrows.

If one watches the exposed heart of a dog, cat, or rabbit, it is seen to execute two movements in systole: (1) the general contraction affecting chiefly the transverse diameter of the heart, and (2) a twisting about of the apex from left to right and forwards. This torsion of the apex is the resultant of the several lines of traction exerted by the musculature of the right and left ventricles upon the base of the aorta and pulmonary artery, and modified by the pivoting of the heart against the vertebral column and by the shifting of its centre of gravity owing to variation in its liquid content. The tendency of this movement is to push the apex of the left ventricle against the chest wall, while the left wall of the left ventricle and the right (anterior) wall of the right ventricle move inwards toward the septum. Wherever in man these walls are in contact with the chest wall these inward movements give rise to retraction of the interspaces above them. The surface of the thin-walled right ventricle moreover is actually pulled inwards during systole, so that there may actually be an indentation of its surface which still further contributes to the systolic retraction.

Protrusions and Retractions.—A variety of protrusions and retractions of the interspaces may be seen to occur with each cardiac contraction.

Graphic records of the impulse have been taken by means of the polygraphs described above, the receiving funnel being placed over the area of pulsation exactly as for a jugular or carotid tracing. Tracings can be made either with a rubber-

covered spring tambour like that used for the carotid, or with an open funnel: the former exerting pressure upon the apex, the latter merely recording the compression or rarefaction of the air in the funnel due to the impulse.

The writer also finds that a funnel, made from a soft rubber stethoscope tip stoppered tightly with a perforated rubber stopper penetrated by a glass tube and bearing a rubber tip, is very satisfactory (Fig. 86).

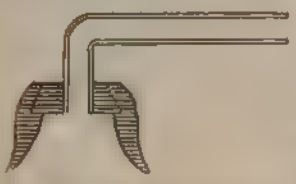

Fig. 86. Rubber funnel for cardiographic tracings.

The movements seen may be divided as:

(1) Lifting of the entire precordium, which results, especially in flat-chested individuals or in those with very large hearts, from the systolic erection of the heart as a whole as it pivots against the vertebral column behind and pushes against the chest wall in front.

This is usually seen in hearts which from any cause whatever are beating heavily, though it is most marked over large hearts.

(2) The normal type of apex beat consists of a large protrusion synchronous with and lasting throughout the duration of ventricular systole (Fig. 88, I, *s–d*), usually preceded by a small presystolic wavelet (*a–s*), due

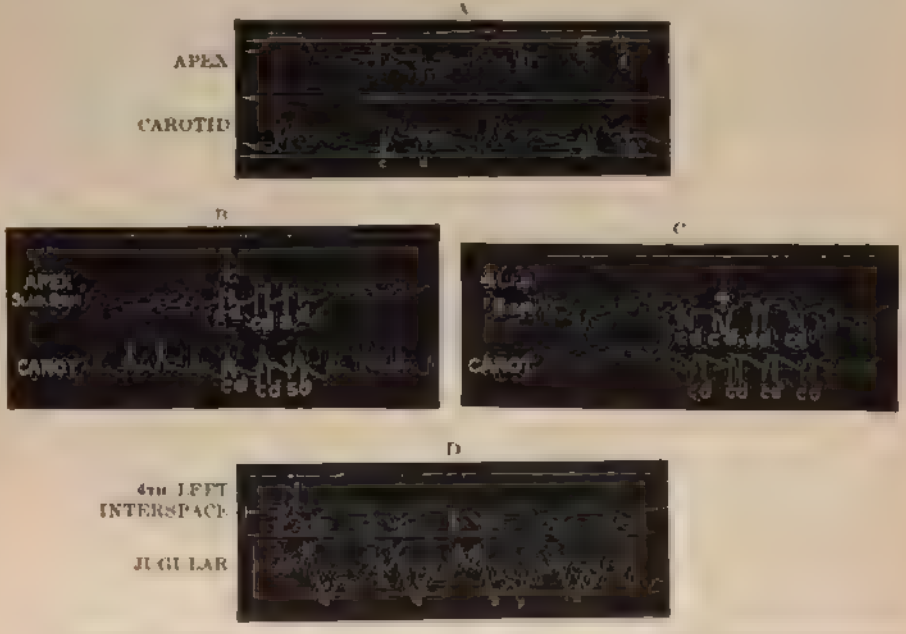

Fig. 87. A, tracing from the apex impulse and carotid artery. *c*, time of carotid wave. *d*, time of dicrotic notch. Upper line gives the time in ¼ sec. B, cardiogram obtained over a normal apex. C and D, cardiogram over the fourth left interspace 5 cm. from costal margin, systolic retraction. From the same individual as B.

to systole of the auricles. The large ventricular wave is followed by a fall in early diastole, coincident with the fall in intraventricular pressure. After this fall there is sometimes a small upstroke of the lever (passive protrusion of the apex by the inrushing blood) which may terminate in a small protodiastolic wavelet (*p*). This protodiastolic wavelet corresponds

to the shoulder upon the cardiac plethysmogram at the end of ventricular filling (page 9), and is particularly marked in cases in which a third heart sound can be heard (Thayer).

In cases with hypertrophy of the left ventricle the protrusion is usually very forcible and heaving throughout systole —dome-like protrusion, choc en dome (Bard).

Occasionally, however, especially when there is some hypertrophy of the right ventricle, the systolic protrusion may not last throughout ventricular systole, but may be represented by only a momentary protrusion, followed by a retraction during mid-systole (Fig. 88, III). Such a beat, which really represents the algebraic sum of the systolic protrusion over the left ventricle and the systolic retraction over the right, may be

Fig. 88.—Various forms of apex tracings. I. Normal, showing presystolic auricular wave *a*, systolic plateau *s-d*, and the curve of ventricular filling *d-p*, ending in the protodiastolic wavelet *p*. II. Normal apex beat, showing only systolic elevation. III. "Mixed" type of impulse, showing an elevation followed by a retraction during the period of systole. IV. Systolic retraction. Apex formed by the right ventricle. V. Mixed type of apex beat, showing protrusion during auricular systole and retraction during systole of the ventricle.

termed a "mixed" type of apex beat. In other mixed types there may be protrusion during auricular systole (presystolic protrusion) followed by retraction during systole of the ventricle (systolic retraction). The right ventricle plays the leading rôle in the production of such an impulse.

(3) Systolic retractions over the entire right ventricle (third, fourth, fifth left interspaces between the parasternal line and sternal margin) when this chamber is hypertrophied or contracting strongly, sometimes also in second left interspace (Mackenzie). Occasionally, especially in cases of mitral stenosis, the presence of a systolic retraction of the interspaces over the right ventricle and a systolic protrusion over the apex gives the cardiac impulse the wavy appearance of a peristalsis. In reality, however, the two movements are synchronous. It is not a peristalsis but a see-saw movement.

(4) Systolic impulse in the second right interspace in aortic insufficiency.

(5) Systolic impulse in the second left interspace (pulmonic area) in pulmonary insufficiency or vigorous contraction of the right ventricle.

(6) Systolic retraction at the apex in adherent pericardium or when the apex is formed by an hypertrophied right ventricle.

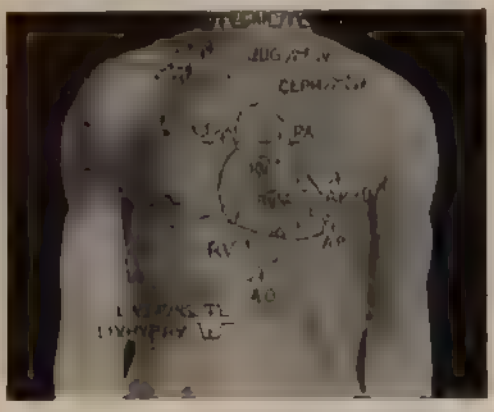

FIG. 89.—Areas of pulsation and retraction. +, protrusion; −, retraction. *CAR* carotid artery. *JUG* jugular vein. *CEPH* cephalic vein. *AO*, aorta. *PA*, pulmonary artery. *RV*, right ventricle. *APHD*, apex with high diaphragm. *AP* apex. *LIV TRINS*, liver pulsation in tricuspid insufficiency. *LIV HYP RV*, liver retraction with hypertrophy of right ventricle.

(7) Systolic retractions in the interspaces beyond the apex (left axilla) due to negative pressure over those areas of lung produced by contraction of a very large heart or to pleuropericardial adhesions.

(8) Retraction of the xiphoid process or ribs from traction of costopericardial adhesions during systole (Broadbent's sign).

(9) Systolic impulses in various abnormal sites due to aneurisms, tumors, or tortuous sclerotic arteries.

PALPATION.

Palpation of the precordium and thorax is undertaken with a view to determine, (1) the force of the apex impulse; (2) the presence and force of any diffuse heave; (3) the intensity of the shock accompanying the heart sounds, (4) the presence and distribution of "thrills"; (5) the presence, distribution, and character of other pulsations.

FIG. 90.—Eddies producing thrills as illustrated by a stream of water. Arrows show lines of force. The large arrow indicates the pressure at the point of palpation.

Thrills.—Corrigan (1837) and, later, Marey showed that thrills may be imitated by producing a constriction in a rubber tube attached to a water faucet. It will be seen that this causes the stream to assume a corkscrew form, giving rise to eddies, twists, and nodes below the constriction. These tend to produce zones of constriction and dilatation in the tube itself and thus set it into vibrations which are palpable as thrills and audible as murmurs. Above the constriction there are no eddies, hence neither thrills nor murmurs.

The thrill is best transmitted in the direction of the stream producing it. It disappears when the constriction becomes too great or the pressure falls too low, and increases with the force of the stream (blood-pressure).

PERCUSSION.

It is of the greatest importance to determine the exact outline of the heart. As has been seen, this is done most accurately by means of the orthodiagraph (page 85), but under ordinary clinical conditions this is not available and the cardiac area is outlined by percussion.

In determining the area of cardiac dulness it is important to map out, (1) the area of cardiac dulness, or, more accurately, the relative cardiac dulness; (2) the area of absolute dulness or cardiac flatness.

RELATIVE CARDIAC DULNESS.

In mapping out the area of relative cardiac dulness it is important to begin percussion as far away from the heart as possible, and then to approach the heart, marking the points at which the very first change of note can be recognized as the heart is approached. In this way one obtains an absolutely resonant note as long as the plessimeter finger is over lung tissue, and a sharp contrast to this as soon as one percusses over the borders of the heart; whereas, if one were to begin percussion over the heart and percuss outward there would be a gradual change of note, becoming more and more resonant, until it finally faded into the perfect resonance over the lung

Fig. 91. Goldscheider's orthopercussion

Choice of Methods.—In outlining the cardiac area one has the choice of several methods

(1) Direct or immediate percussion by tapping the chest wall directly with the finger-tips of one hand

(2) Heavy indirect or mediate percussion.

(3) Medium-light percussion

(4) Lightest audible percussion (threshold percussion of Ewald, Goldscheider Curschmann and Schlayer).

(5) Palpatory percussion (Ebstein by note too low to be heard at all.

(6) Orthopercussion (Goldscheider) (Fig. 91), distal phalanx of the plessimeter finger held perpendicular to the chest wall

(7) Instrumental percussion with a mechanical plessimeter, the blow being struck by either the finger or a hammer

In selecting the method of percussion it should be borne in mind that, though bodies near the chest wall on either side of the line of percussion stroke may tend to damp the vibration of the lung and impair the resonance of the note, this tendency is greater for heavy percussion and loud resonance than with light strokes which set only small areas of lung in vibration. Moreover, it is a well-known law of sense-perception that the softer the initial sound the easier it is to detect variations in it Indeed, de la Camp goes so far as to recommend light direct percussion through a single layer of blanket laid upon the chest as the most accurate method of outlining the cardiac dulness.

Moritz, Dietlen, de la Camp, Goldscheider, Curschmann and Schlayer, and a number of other writers have compared outlines made by the various methods of percussion in hundreds of cases with those obtained by the orthodiagraph, while Simon has marked out his outlines by percussion upon the intact cadaver with pins and then tested his accuracy upon opening up the thorax. All these observers are unanimous in advocating very light percussion for outlining the left border of the heart, but Moritz prefers a rather heavy palpatory percussion for the right border.

Moreover, the sensations which percussion imparts to the finger are more delicately graded for a light stroke than for a heavy one, since the

pressure of a heavy blow somewhat dulls the sensibility of the finger-tips, and in this way also a light stroke is more satisfactory. The oft-made claim that a light stroke does not penetrate deep enough for mapping out the right border of the heart, though seeming plausible, is not warranted by experience. On the contrary, the writer has observed that those clinicians who rarely make out at all the area of cardiac dulness which lies to the right of the midline were usually those who used heavy percussion.

Avoidable Errors in Percussion.—The exact method used is a matter of individual preference and practice. The essentials for all forms are (1) a loose wrist[1] loosely held finger-joints, and a short sharp blow with immediate elastic recoil, (2) firm pressure of the plessimeter finger against the chest wall especially in the inter-spaces. In the writer's experience the important point is not the method used but the care in discriminating the first slight differences in note and sensation. The errors of percussion so frequent among students and even experienced physicians are far more frequently *due to inability to detect differences in note than to inability to elicit them*. This inability to detect slight differences was due in most cases to a preconceived notion as to the intensity of change obtainable. The observer usually expected a greater change and permitted his ear to neglect the lesser, although once his attention was called he was perfectly able to detect it.

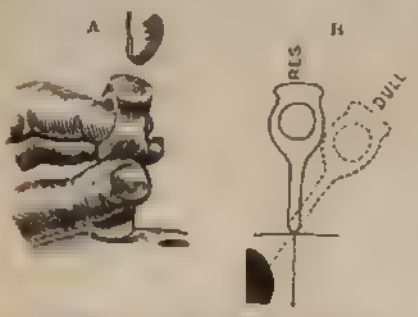

FIG. 92.—Percussion with the orthoplessimeter. A, J. O. Hirschfelder's orthoplessimeter and its mode of application. B, Supposed line of transmission of the percussion impulse from the orthoplessimeter. *RES*, resonant percussion note.

Special Methods of Percussion.—The method of choice varies somewhat with the purpose. For ordinary purposes very light direct percussion is quite satisfactory, or ordinary threshhold percussion with barely audible note. Where *accuracy* is important, as in determining the mobility of the heart or of the lung borders, Goldscheider's *orthopercussion* or J. O. Hirschfelder's *orthoplessimeter* is preferable.

Goldscheider believed that orthopercussion was so delicate that dulness was given only by bodies directly in the axis of the plessimeter phalanx and that in this way the plane of an oblique surface could be detected, but experience shows that this is rarely possible. It succeeds much more frequently when the orthoplessimeter (Fig. 92) is used, so that a resonant note may be obtained when the shaft is pointed parallel to the heart surface, a dull note when it is pointed toward the heart.

Unavoidable Errors in Percussion Outlines.—In outlining the heart by percussion the right and left borders present different problems. The right border is situated deeply and recedes at once from the chest wall, so that it represents the first point at which dulness could be obtained. The left border is superficial and convex and the convexity sometimes follows the curve of the ribs in the left axilla. Accordingly it may happen that in round narrow chests or in persons with large hearts the left ventricle may almost fill the left half of the thorax. The curve of the ribs follows the wall

[1] Some persons are possessed of a loose wrist at once, others acquire it only after long practice. For the latter the writer recommends the following exercise practised two to five minutes daily. Hold the wrist as loosely as possible, then vibrate the forearm very rapidly to and fro from the elbow until the hand shakes about like a tail upon the loose wrist too fast for the eye to follow its movements. The improvement in percussion following this exercise is very gratifying.

of the left ventricle and the latter may remain near the chest wall throughout the axilla. The outer border of dulness may thus be obtained not over the apex but over the posterior wall of the left ventricle. In persons with narrow chests or much enlarged hearts the area of dulness (Fig. 93, P—P) extends around the heart and not merely across the transverse diameter (O—O). The transverse diameter (O—O) corresponds accurately to the point mapped out with the orthodiagraph. Accordingly there may be a discrepancy of several centimetres between the percussion and orthodiagraph estimations of the distance from the midline to the left border. In broad flat chests where, beyond the apex, the left ventricle recedes from the chest wall, this discrepancy does not occur; and the findings by percussion and by X-ray coincide closely. In a very large series of cases Moritz found his percussion (light percussion for the right border, threshold percussion for the left) to be correct for the right border in 86 per cent., for the left in 70 per cent.

Fig. 93.—Diagram to show the cause of unavoidable error in percussion of the cardiac outlines. *P*—*P*, outline on percussion; *O*—*O* orthodiagraph outline.

DIAMETER OF THE CARDIAC AREA.

In mapping out the area of cardiac dulness the position of the apex is given designating the level of rib or interspace during quiet respiration, and the number of centimetres to the left of the midline (Figs. 84 and 94) (ML). The level of upper border at the left sternal margin is given and also the distance to the right of the midline (MR) in the fourth right interspace. The acuteness or obtuseness of the angle formed between the hepatic and the cardiac dulness (cardiohepatic angle, angle of Ebstein) is also noted. In addition to this Moritz and Dietlen call attention to the importance of recording the two diagonal diameters of the heart (longitudinal, L, from apex to the aortic angle of the dulness, and transverse, Q, from the cardiohepatic angle to the upper left border, as shown in Fig. 84). Normal figures for these conjugates according to Dietlen are

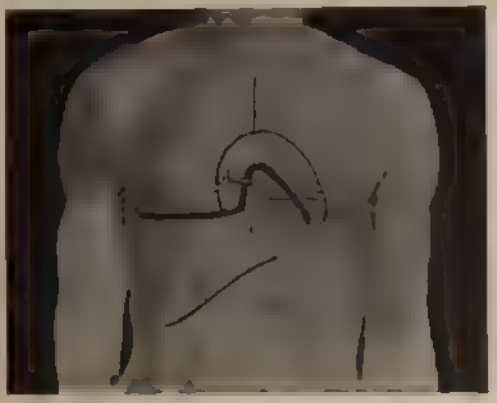

Fig. 94.—Areas of cardiac dulness and flatness in a normal man. The outer broad line represents cardiac dulness, the inner heavy line represents cardiac flatness.

Height of individual		Men					Women				
Cm	Feet and In	MR Cm.	ML Cm	L Cm	Q Cm.	Cardiac area Qcm.	MR Cm.	MI Cm.	L Cm.	Q Cm	Cardiac area. Qcm.
145 154	4.7—5.	3 5	7 9	12 5	9 7	95	3 5	8 1	12 7	9 4	93
155 164	5 1 5 5	4.1	8 7	13 8	9 9	109	3.5	8 4	13.2	9 7	101
165 174	5 5 5 9	4 2	8 8	14 1	10 3	116	3.8	8.5	13.4	9 9	105
175 187	5 9—6 2	4 4	9 1	14 8	10 7	127					

Dulness in Children.—In children the heart is proportionately larger and lies more transversely than in adults. The apex is usually in the fourth interspace lateral from the nipple. Veith has shown that the cardiac shadow in children extends exactly twice as far to the left as to the right of the midline (ML . MR 2 . 1).

Changes in the Relative Dulness. The relative proportions of the various conjugates undergo quite typical changes in various forms of heart disease. In weakening of the right heart, in tricuspid insufficiency, and tricuspid stenosis the conjugate MR is increased (dulness increased to the right); in hypertrophy of the left ventricle and in mitral insufficiency, dulness increases to the left (MR increased), while in the latter condition as well as in mitral stenosis the oblique transverse diameter (Q) is increased. In aortic disease there is lengthening of the long axis (L).

Fig. 95. Cardiac outlines in a child of nine years.

CARDIAC FLATNESS

The area of absolute dulness or cardiac flatness represents the portion of the heart which is not covered by lung (Figs. 93 and 94). It forms a triangle extending from the fourth rib above to within 2 3 cm of the apex, or even just to the latter in the fifth left interspace.

It is best mapped out by very light percussion, beginning over in the fifth left interspace at the left sternal margin, percussing lateralward and upward, passing from the absolute flatness to the area of impaired resonance instead of in the opposite direction).

Variations in the Area of Flatness.—In the primitive mammals (dog, cat, rabbit) the heart does not lie in close apposition to the chest wall, but is slung rather loosely between the folds of the mediastinum and completely covered by lung. There is no area of flatness. This same condition is met with in many otherwise normal persons, especially in the long flat-chested, and in those who have extremely movable hearts or general mobility of all the viscera (visceroptosis, enteroptosis, page 598).

Entire absence of cardiac flatness is also found in the exact opposite type of chest, in the barrel-chest patients with emphysema, in

whom the exaggerated efforts at inspiration have caused the lungs to be sucked in gradually between the heart and the chest wall.

On the other hand, the area of cardiac flatness is often enlarged in persons with flat, rhachitic, or tuberculous chests. In hypertrophy of the right ventricle the area of flatness is enlarged and the right border becomes oblique, extending downward to the right margin of the sternum, often interrupted by step-like protrusions (Kroenig). In pericardial effusion it extends well into the fifth right interspace.

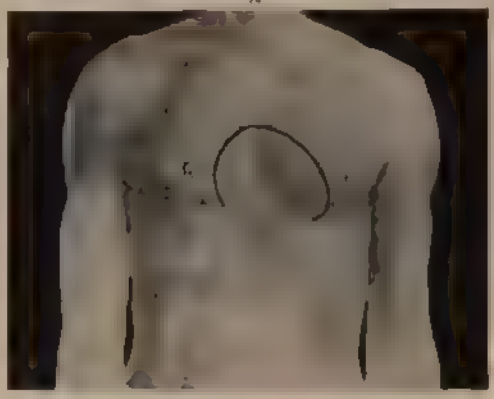

Fig. 96. Diagrams illustrating the movements of the normal heart on change of posture from side to side (A) and the various phases of respiration (B). Solid black line, normal cardiac outline in quiet breathing; dotted line, R, cardiac outline with patient lying on right side; broken line, L, cardiac outline with patient lying on left side; EXP, horizontal shading, outline in expiration; INSP, vertical shading, cardiac outline in inspiration. The movements shown in these figures represent the upper limits of normal mobility.

Changes in Size of the Heart.—As seen in the investigations upon cardiac volume, the size of the heart, and hence the area of cardiac dulness, is subject to a physiological increase when the heart is slow and decrease in size when it is rapid (Henderson, see page 9). This decrease in size is especially noticeable in certain cases with rapid hearts, like paroxysmal tachycardia when there is no heart failure nor vasodilation (Hoffmann, Dietlen). An increase in size may be associated with a slow pulse (see page 9 and Fig. 12), hypertrophy of the heart, or with a pathological dilatation. The physiological condition should first be considered before assuming the pathological.

Changes in Position of the Heart.—(1) Upon changes in posture. Normally changes in posture are accompanied by considerable changes in the position of the heart. The apex may move 3–5 cm. when the patient turns from one side to the other, always moving towards the side which is lower. On standing a similar but less marked change occurs. Moritz, and, later, Dietlen have shown that the area of the cardiac shadow is from ten to thirty per cent. smaller on standing than on lying down. The latter observer confirms Erlanger and Hooker in stating that the pulse-pressure, and hence the systolic output of the ventricles, diminishes correspondingly.

The diminished filling of the heart is due also to the fact that the pressure under which the blood enters it in diastole (venous pressure) is lower upon standing than upon lying down.

In some people extreme mobility (6–8 cm.) of the apex is found (wandering heart)—a condition often associated with cardiac neurasthenia and palpitation, and even paroxysmal tachycardia. Changes in position of the diaphragm, upon expiration, inspiration, or intestinal flatulence, also affect the position of the heart, especially upon standing; so that in expiration or flatulence the apex is pushed up and the heart lies more transversely, while in inspiration the apex falls and the heart lies more nearly in the long axis of the body (Fig. 96). As can be readily shown with Henderson's cardiometer, the former position interferes with the cardiac filling and hinders the circulation, while the latter position facilitates both. The amount of change of position of the apex is normally about 1–2 cm.

AUSCULTATION.

CHARACTER AND TIME OF THE HEART SOUNDS.

The beat of the heart is accompanied by two definite sounds ordinarily likened to the syllables "lub-dub" or "tă tă," the first sound accompanying systole, the second occurring just at the beginning of diastole.

Graphic Records.—The exact period of the cardiac cycle to which they correspond was first investigated by Donders (1856), who marked the onset of the sounds by tapping upon a receiving tambour the instant he heard sound and recording this signal upon a drum while simultaneously recording the cardiogram. This method was subsequently developed by Martius and has furnished some interesting information, but even in regular pulses the results are very fallible and the method cannot be used at all when the pulse-rate is irregular.

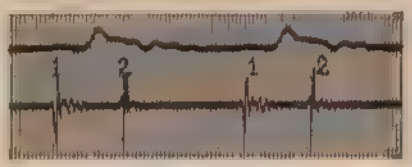

FIG. 97.—Graphic records of the heart sounds. (Kindness of Prof. Einthoven.) Each vertical division represents .02 sec.

During the past fifteen years several methods of recording the heart sounds graphically have been devised (Einthoven and Geluk, Huerthle Holowinski). Einthoven, Flohil, and Battaerd place a microphone over the heart, connect the microphone with the thread galvanometer, and photograph the movements of the latter.

O. Frank attaches the tube of a stethoscope to a tambour over which there is stretched a delicate condom membrane bearing a small mirror. A beam of light is thrown upon this mirror, and its movements, concident with the sound waves in the stethoscope, are recorded photographically.

A still more delicate method is that of Weiss and Joachim. Instead of the condom membrane used by Frank, these investigators register the vibrations of a soap bubble film blown over their receiving tambour. To prevent bursting this is kept in a moist chamber of glass. The vibrations are magnified by means of a small *L* of glass capillary which rests upon the film. The movements of the shadow cast by the end of the *L* are recorded photographically. Weiss and Joachim's results are at least as good as those

of Einthoven. Their records are similar and quite as delicate, and add much to our knowledge of heart sounds. Moreover, they are able actually to synthetize and reproduce these sounds by transferring their curves to zinc strips which are rotated upon a drum and set a stile in motion. The vibration gives rise to sounds which they state have been identified by other clinicians with those of the cases recorded.

Several methods of recording the heart sounds by vibrations of a gas flame (Mazbe, Roos) have been only moderately successful, and though simple are not as satisfactory as the photographic methods.

Clinical Diagram for Heart Sounds.—In many text-books the heart sounds are represented graphically in various ways, but it seems to the writer that the best is to indicate the occurrence of the sounds directly upon a simple diagram which indicates the relation to the auricular and ventricular contractions, as shown in Fig. 98.[1]

FIG. 98.—Diagram for representing the heart sounds in clinical notes. Upper curve represents the events of the cardiac cycle, the small auricular contraction followed by the larger ventricular contraction. Lower line represents the heart sounds. True heart sounds are represented by solidly shaded blocks, whose height indicates their intensity and whose breadth indicates their duration.

CAUSES OF THE HEART SOUNDS.

First Sound.—Harvey states that "when there is the delivery of a quantity of blood from the veins to the arteries, a pulse takes place which can be heard within the chest." Laennec (1819) was the first to describe the character of the sounds. He regarded the first sound as due to ventricular systole, though he thought the second to be due to the contraction of the auricle. In 1836, C. J. B. Williams and a committee of the British Medical Association investigated the heart sounds experimentally. He believed that the first sound was largely of muscular origin, like the contraction sound of skeletal muscles, because it could be heard upon the excised heart even when the auriculoventricular valves were held open with the fingers, but the second sound could not be heard unless the aortic or pulmonic valves closed. This view was substantiated by Ludwig and Dogiel; but Sibson and Broadbent found that in the exposed heart of the ass the first sound begins with a sort of rumble, which disappears when the blood flow is shut off by tying the venæ cavæ. This rumble they ascribe to the movement of the auriculoventricular valves.

Graphic records of the heart sounds by Einthoven, Flohil, and Battaerd have shown that the first sound in man begins at the beginning of ventricular systole and lasts 07 to 10 sec. It is loudest at its very beginning, is decrescendo in character, and is almost completed before the aortic valves open, i.e., before the heart has begun to pump blood into the aorta. The first sound is followed

[1] Thus in cases of mitral stenosis (see page 348) the first sound may be short and tapping in character, though tracings show the systole to be of duration no less than that met with in the absence of tapping character (Hirschfelder).

by the short pause, which usually lasts .15 to .25 sec., and which is then followed by the second sound. Einthoven's results have been confirmed in man by the records of Weiss and Joachim, Hess and Frank, as well as by Prof. Barker, Dr Bond, and the writer. In the dog, R. H Kahn has shown that the duration of the first sound is exactly coincident with the period during which the intraventricular pressure is rising, while the duration of the short pause is exactly coincident with the systolic plateau.

Sahli and other clinical observers believe that the first sound at the aortic area begins later than that at the apex and is due to the rush of blood from the ventricle into the aorta, but graphic records seem to indicate that the sounds in the two areas are synchronous, and begin before the aortic valves open.

However, the first sound heard on listening in the suprasternal notch is often split; and it is possible that the latter portion of this sound is due to just such a forcible distention of the aorta.

The valvular element of the sound is probably brought about when the valves are thrown into tension by the ventricular systole. The normal valves give no sound at all when they open spontaneously.[1] There is no evidence to indicate that the normal sound is brought about to any extent by eddy currents as are thrills or murmurs, nor does systole of the auricles produce any portion of the normal first sound (Einthoven).

Hess and Frank believe that the movement of the heart within the chest and perhaps against the chest wall (systolic erection) may be an important factor in the production of the first sound. This might explain why the heart sounds are occasionally inaudible in emphysematous persons in whom the organ is separated from the chest wall by a layer of lung. On the other hand, this factor is shown to play only a minor rôle by the fact that the first sound may be heard in its normal intensity in the exposed and even the suspended dog's heart.

Second Sound. The second sound has been shown by C J B. Williams and the British Commission to accompany the closure of the aortic and pulmonary valves, to be modified when these valves are injured, and to disappear when they are held against the vessel wall It lasts about .05 second It is loudest when the blood-pressure is high, when the valves are thicker and more rigid than normally, or when the vessel walls are more elastic than usual, the intensity varying at different times of life and under pathological conditions.

METHODS OF AUSCULTATION

Monaural Stethoscope. — The monaural stethoscope, introduced by Laennec, is a simple wooden tube surmounted by a flat disk acting as an ear-piece and resonator The tube is pressed against the chest and the ear laid upon the disk so that the observer receives at once the sound and the thrill in the wood transmitted directly. Obviously this method accentuates the notes of low pitch which are nearest to the essential tone of the instrument (and constitute most of the normal sounds) as well as those

[1] Both the valves and the cardiac walls are at that time extremely lax and the valvular opening is almost equal to the diameter of the ventricular chamber

of relative loudness, which cause it to vibrate mechanically. Hence it is particularly adapted to the detection of presystolic and other rumbling murmurs, and is the method used almost exclusively outside of the United States.

Binaural Stethoscope.—In the United States the binaural stethoscope is in more general use. This consists essentially of a small receiving bell which is placed upon the chest wall, and from which two tubes lead off to small rubber ear-pieces which fit tightly into the external auditory meatus. The most important essentials in these three forms are, (1) a bell composed of various materials—ivory, wood, celluloid, or hard rubber—provided with a sufficiently large air space at the tip (Emerson); (2) ear-pieces perfectly fitting the ear of the individual. It is safe to say that more errors of auscultation result from poorly fitting ear-pieces than from real inefficiency on the part of the listener. (3) In stethoscopes in which the ear-pieces are held in the ears by a spring this should not exert excessive pressure lest it produce sounds within the ear from the pressure on the drum.

Fig. 99.—Choice of stethoscope bells.

There are three main forms of binaural stethoscope: (1) those with rigid tubes (Gannett's), (2) those with soft rubber tubes, (3) those with soft rubber tubes, flat bells, and a small elastic disk of metal or celluloid to act as a resonator (Bowles). Of these three forms it may be said that the rigid tubes certainly convey the sounds somewhat better but this is often more than compensated for by the better fitting of the ear-pieces in stethoscopes with soft rubber tubes. In stethoscopes with disks certain sound waves, and particularly those of high pitch (soft blowing murmurs), are accentuated, while other sounds may be relatively suppressed. Moreover, any movement of skin or hair over the disk may give rise to a sound simulating a friction, and this source of error must be carefully excluded. Hair should be moistened, and a small bell should be used with perfect approximation to the skin throughout its circumference.

Alteration of Sounds by Pressure. Emerson has shown that many murmurs, especially presystolic and snapping sounds, are diminished or obliterated by pressure with the stethoscope, while certain others are intensified by pressure, and that this is dependent upon the pitch of the sound and not upon the site of its production. It is therefore important for the observer to listen carefully, first with the lightest possible pressure upon the stethoscope and then with gradually increasing pressure. He should do this consciously and as a matter of routine, rather than allow such sounds to escape him or stumble upon them by accident.

Moreover, since the monaural and binaural stethoscopes each intensify different sounds, both should be used in any important or dubious case before the examination is concluded.

Graphic Methods.—As stated above, the most accurate and reliable information which has thus far been obtained is that obtained by means of the recording microphone. Unfortunately, however, all the methods

thus far devised have been too cumbersome for the bedside or even for routine hospital use. They are of value only in exceptional cases for research, but there is no doubt that the future of scientific auscultation lies in this field.

"VALVULAR AREAS" IN AUSCULTATION.

The various cardiac sounds are best heard over certain definite locations corresponding more or less to the structures in which they arise, but particularly to the course of the blood current and to their mode of origin (Fig. 100). Thus the sounds produced in the left ventricle are best heard at the apex; those produced at the aortic orifice, though produced behind the sternum, are heard just to the right of it in the second interspace; the pulmonary sounds are carried to the second left interspace at the sternal margin, while the sounds from the right ventricle are heard over the entire body of the sternum, over the greater part of the area of absolute dulness, and over the base of the ensiform cartilage. Abnormal sounds, murmurs, etc., have, however, a different distribution which will be discussed later.

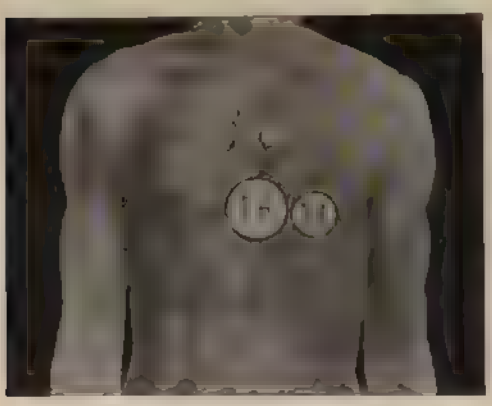

Fig. 100.—The "valvular areas."

Normally the first sound at the apex and everywhere else below the third rib is louder than the second sound. It is also of longer duration than the latter (.08 second as compared to .05). Over the aortic and pulmonic areas it becomes somewhat fainter, begins a trifle later, and is of longer duration than over the apex. The second sound is then louder than the first.

The second sound at the second left interspace (pulmonic second) is usually louder than that over the second right (aortic second) up to the age of 25 to 30, when the latter becomes the louder (Cabot).[1] This varies greatly in different individuals. Mere changes in blood-pressure are not sufficient to account for all these conditions, since the pressure in the pulmonary artery is never more than half that in the aorta, but proximity to the sternum, greater elasticity of the walls, etc., combine to bring about the relative loudness of the second pulmonic sound, and therefore any further increase in pressure in either artery alters the relation of the two sounds to each other, increased pulmonary pressure increasing the pulmonic second, increased general blood-pressure increasing the second aortic, etc. The progressive thickening of the aortic semilunar valves after the age of 30 also contributes to the intensity of the sound.

Other Sites for Auscultation.—Bou-Teissier has also recommended auscultation in the *suprasternal notch*, pressing the bell of the stethoscope as far down behind the manubrium as possible. In this way he states that he can hear aortic diastolic murmurs

[1] Directly over the exposed aorta the sound is louder than over the exposed pulmonary artery (Thayer).

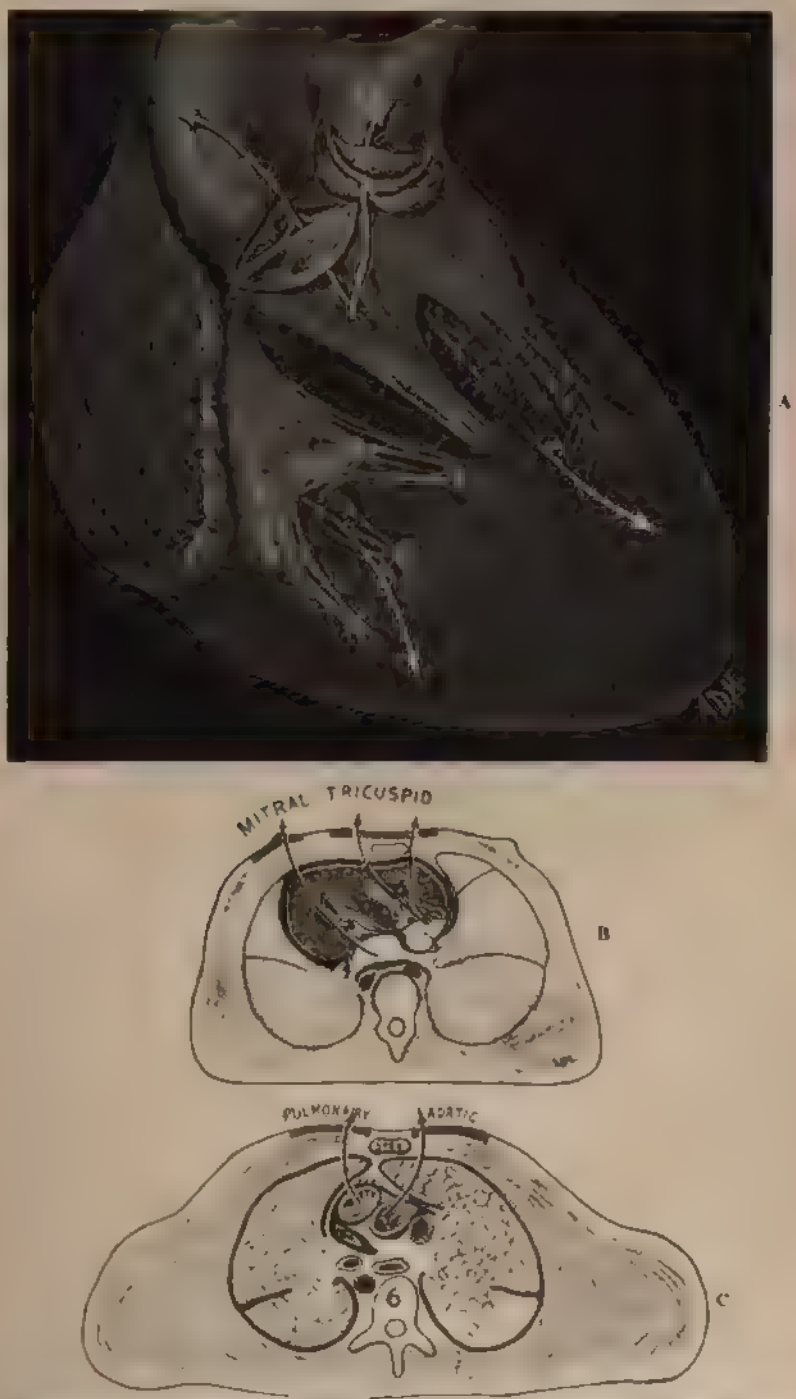

FIG. 101.—The propagation of the heart sounds from valves to chest wall. A. Course of the sound waves within the heart. B. Propagation of the heart sounds at the level of the second interspaces. C. Propagation of the sounds at the level of the fourth and fifth interspaces.

not otherwise audible. He thinks that he is also better able to distinguish the character of aortic systolic murmurs. The method has never gained general usage, and the writer is unable to find in it any of the advantages claimed by Boy-Teissier. The chief value of suprasternal auscultation is found in persons whose heart sounds are feeble or inaudible over the precordium. It must be borne in mind, however, that the mitral murmurs are not well transmitted to this region, and that the first sound heard there is frequently reduplicated or split.

Another form of auscultation not in general use is the *auscultation through the stomach-tube*, introduced as for a tracing from the left auricle. This method, first used by A. Hoffmann in 1892 has been revived by Gerhartz, but, though it might throw some light upon the nature of an occasional mitral murmur, it is in general difficult and very inconvenient to carry out; and in many cases at least the murmurs are no better heard than over the chest wall. Nevertheless where it is important to know whether a murmur is conducted back into the left auricle, a positive finding by this would be conclusive.

EMBRYOCARDIA.

Ordinarily the diastolic pause between sounds is longer than the systolic period, and the interval between the second sound of one cycle and the first sound of the next is longer than the interval between the first and second sounds of the same cycle. However, when the heart-rate is very rapid, the diastolic pause may become shortened to about the same interval as that between the first and second sounds (*long pause = short pause*), so that the sounds succeed one another at uniform inter-

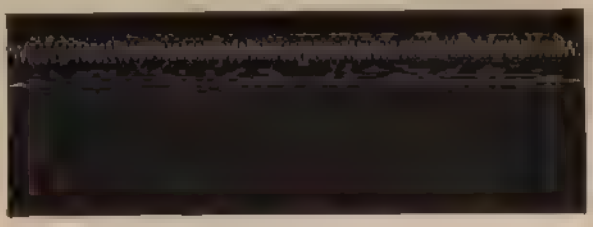

FIG. 102. Graphic records of the fetal heart sounds. (After Weiss and Joachim.)

vals like the ticking of a clock. This rhythm is heard normally over the fetal heart and hence has been termed embryocardia or fetal rhythm. It also occurs in adults when the rate is very rapid (120 and over), and hence under conditions in which the heart is under an abnormal strain (see page 227), as in fevers with high temperature, acute heart failure, and acute overwork of a chronically diseased heart, also in cases of paroxysmal tachycardia and allied conditions. Its absolute significance is simply that of the rapid heart-rate to which it corresponds.

ACCESSORY HEART SOUNDS.

REDUPLICATED SOUNDS AND GALLOP RHYTHMS.

Reduplicated Sounds.—Occasionally one or the other of the two normal heart sounds is replaced by two clear sounds, or, in other words, there is a reduplication. This reduplication may occupy the place of either the first or the second sound, and, as already noted by Skoda, it may seem to be

due to, (1) splitting of the normal sounds into two distinct portions, or (2) pressure of an accessory sound besides the normal sound, being in the latter case presystolic (before the first sound), protodiastolic (shortly after the second sound), or mesodiastolic (in mid-diastole). The relation of groups 1 and 2 to one another and to the cardiac cycle is shown in Fig. 103.

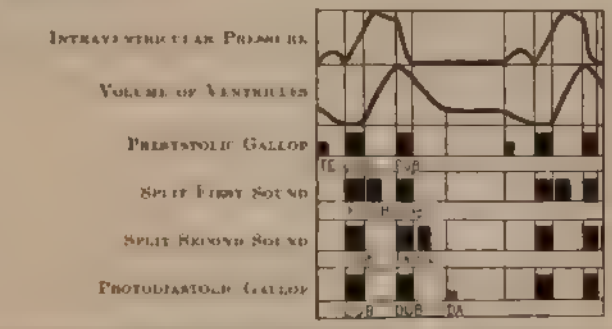

Fig. 103.—Diagram illustrating the split sounds and gallop rhythms and their phonetic equivalents.

As to the causation of these abnormal sounds, little definite is known. A great deal of the indefiniteness which permeates the enormous literature upon the subject is due to the failure of the writers to distinguish clearly between the different forms with which they are dealing. The presystolic and protodiastolic forms are grouped under one head regardless of their relations to mechanism or etiology. It is mainly due to the writers of the French school under the leadership of Potain that the differentiation has reached even its present stage of development. L. Bard, of Geneva, has recently given an excellent analysis of the subject from this stand-point.

According to Bard, the two main groups of accessory sounds are:

(1) The presystolic gallop reduplication (ta-ta-tat) or ta ta tat, to which the term gallop rhythm should be limited; most commonly met in nephritics with cardiac hypertrophy and in other heavily beating hearts.

(2) The protodiastolic sound ta ta ta (dub-dub-dub), called by Bouillaud bruit de rappel,—"sound of recall" or "diastolic echo,"—frequently heard at the apex in mitral stenosis. Bard thinks that the above mentioned accessory sounds are to be regarded as merely the exaggeration of vibrations normally present but normally inaudible.

Split Sounds.—The sounds (tlat-tat; tat-tatl) are characterized by the absolute similarity and short interval between the two portions, and may be due either to slight asynchronism of the two ventricles (C. J. B. Williams, 1836, Skoda, Gibson, 1874) or slight separation of two parts of the ventricular sound, which are of different origin but ordinarily fused.

As has been seen, the ventricular sound contains both a valvular (auriculoventricular) and a muscular element, and perhaps also an element due to the stretching of the walls of the aorta. Bard thinks that variation in either the muscular or the valvular element might give rise to their separation into two sounds. The question of asynchronism of the two ventricles which arises in this connection is one which was long without an experimental basis, but the recent observations of Stassen, Kraus and Nikolai, and Hewlett indicate the possibility that it may occur clinically. Stassen, in Frederecq's laboratory, has recorded asynchronous contractions of the two ventricles when the latter were recovering from vagus inhibition, and also with ventricular extrasystoles produced during periods of vagus inhibition. The writer has on one occasion heard a split first sound in an animal in which the contractions of both ventricles were being recorded with myocardio-

graphs. The ventricular contractions were slightly asynchronous. In a number of other instances in which no split first sound could be heard the contractions were absolutely synchronous. However, no conclusions are justified from an isolated observation.

The splitting of the first sound is best heard over the base and body of the heart, in contrast to the accessory sounds which are best heard at the apex (see below). As to the splitting of the second sound, this likewise may be due to slight asynchronism of the two ventricles, or to the fact that even without this the semilunar valves may not close at exactly the same instant. It is often possible, by passing the stethoscope along the second right and left interspaces, to determine which second sound lags behind.

It must be added, however, that, as Bard himself states, no accurate knowledge of either the split sounds or the accessory sounds can be gained until they are registered graphically by cardiophonographic methods along with simultaneous venous, arterial, or cardiographic tracings, so that their

FIG. 104.—Graphic record of a split pulmonic second sound. (After Weiss and Joachim.) *PRE.*, presystolic rumble, *SYST.*, systolic murmur, 1, first heart sound, 2 A, B, two parts of split second sound (.04 sec. apart).

exact relation to the cardiac cycle may be determined. So rapid is the sequence of the sounds that in an individual case the differentiation between split and accessory sounds is often difficult.

Reduplication of the First Sound from Pericardial Adhesions.—Reduplication of the first sound is also heard in a number of cases in which old pericardial and pleural adhesions are found at autopsy (Sewall), which may be easily understood to give an abnormal sound in systole. Just how commonly this group occurs has not been determined statistically, but under these conditions it need not signify any disturbance of function.

Presystolic Gallop Rhythm.—As regards the accessory sounds, the great majority of writers take the view suggested by Exchaquet in 1875 and Johnson in 1876 that the first sound of gallop rhythm (presystolic sound) is due to the vigorous systole of the auricle, a view which is further supported by the studies of Kriege and Schmall (1891), Friedrich Muller (1906), G. C. Robinson (1908), and others. According to Muller, Marey believed that the extra sound was produced by the auricle sending blood into a defectively emptied ventricle, a view which has been revived by Sewall. Moreover, the writer has been able to show on the excised heart that when the ventricles are distended under a slight positive pressure the auriculoventricular valves may open along only a small extent of their line of closure. This gives rise to a slight functional stenosis at the point where they actually open, a fact which may account for the audible auricular contraction. Muller considers that the extra tone may be dependent upon a

delay in the time between the auricular and ventricular contraction, possibly due to lowered conductivity in the atrioventricular bundle of His, and when the two contractions are abnormally separated two sounds instead of one are produced. Tracings, however, do not usually show delayed conduction.

All these writers base their views upon the fact that the sound appears to be presystolic in time and that in many cases a well-marked auricular wave may be seen upon the cardiogram at a corresponding point of the cycle. It must be added that this is also seen in many cases in which there is no gallop rhythm and that it seems to be dependent more upon the prominence of the apex impulse in the interspace facilitating the record than it does upon the existence of the sound. However, this wave is often quite as prominent in the curves (protodiastolic sound) in which no presystolic sound was heard as in those used to illustrate the gallop rhythm itself. The proof is therefore insufficient, but that does not mean that the theory is necessarily wrong. It is not at all improbable that the forcible contraction of an overloading auricle may give an audible sound just as it does when forcing blood through a narrowed orifice (presystolic rumble), but this has not yet been proved and will require careful investigation with the cardiophonograph. The possibility of functional mitral stenosis like those mentioned on page 371 must also be borne in mind.

Another explanation for the phenomenon is that the sound occurs during the ventricular systole, as suggested by H. Chauveau, who thought it due to the tension of the auriculoventricular valves. His apex tracings, however, are not carefully timed and might quite as well be interpreted as evidence of the auricular sound.

The numerous reviews of the literature, such as those of Obrastow, Pawinski, Robinson, shed no further light upon the subject.

Clinically, the presystolic gallop rhythm is usually met with in cases with rapid hypertrophied hearts which are under a slight overstrain, as in the classical group of chronic nephritis, chronic cardiac disease, aneurism, cases with arteriosclerosis, exophthalmic goitre, mitral stenosis, and acute fevers. Occasionally it is heard in normal individuals (Krehl). It seems in most cases to accompany slight overwork of the heart, but its mechanical and physiological significance is still not clear.

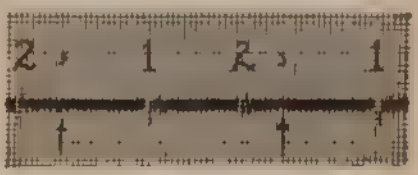

Fig. 105.—Graphic record of the third heart sound. (Kindness of Prof. Einthoven.)

Protodiastolic Gallop Rhythm. Third Heart Sound.—The rôle of the protodiastolic sound (bruit de rappel, diastolic echo) seems to be more definitely established. Though already heard by Bouillaud in 1835, in mitral stenosis, its occurrence was emphasized by Duroziez (1874) and by Sansom (1881), who term it the "opening snap" of the mitral valve, indicating that it was brought about by the opening of the stiffened valve. Barie (1893) and Thayer (1906) called attention to its occurrence in normal individuals. In 1907 the writer observed this sound in a normal individual with a slow and vigorous heart, whose venous pulse showed a peculiar extra wave (Fig. 106, *h*) which follows the inflow of blood into the ventricle (as indicated by the normal *v* wave, Fig. 106, page 108).

The writer also called attention to the fact that this wave bore a close relation to the end of the rapid filling of the heart (or diastole proper) upon the volume curve of the ventricles, and that Henderson had claimed that at this time the mitral valves and tricuspid were closed by the elastic recoil of the heart walls. That this actually takes place and is

dependent upon a high venous pressure can be shown on the dead heart by pouring water into the ventricles from a beaker after the auricles have been cut off in the manner devised

FIG. 106.—Jugular and carotid tracings from a normal individual with a well marked third heart sound showing a large *h* and a smaller presystolic wave *a*. * indicates a small wave in mid-diastole following the A wave occasionally found though perhaps an artefact.

by Baumgarten (1843). If the water is poured from just above the valves they merely float out a little toward the middle of the orifice; if from the height of about 10 cm. they float into apposition; if from 30 cm. above they are left tightly closed when the flow ceases. These observations have been confirmed recently by C. Lian in François-Franck's laboratory. Hirschfelder also suggested that this closure of the valves may be sudden and vigorous enough to cause a sound.[1] The relation of this sound to this portion of diastole seemed quite definite by comparison with a graphic record of this sound made at about the same date by Einthoven (Fig. 105), which shows it to occur 0.18 second after the second sound. This explanation has also been supported by A. G. Gibson and Professor Thayer. The tracings of Robinson, who was investigating the subject from a different stand-point, have also shown the constant presence of the *h* wave upon the venous tracings accompanying this sound. Robinson and Thayer have also shown that it accompanies a wavelet *p* upon the cardiogram in early diastole (Fig. 88, I, page 91), probably due to the filling of the ventricles. They find this wave upon the cardiogram in almost all cases of protodiastolic gallop rhythm and regard it as characteristic of the latter. Thayer has demonstrated that it cannot be an artefact, since it is often both visible and palpable, and hence can often be found by the ordinary simple methods of physical examination. Some venous tracings made from animals by Exner, along with the volume curves of the heart, show that the rise at the foot of the *h* wave occurs at the end of the rapid diastolic filling (Thayer). According to these explanations the sequence of events would be as follows. The end of diastole is marked by the second heart sound and by the fall in the cardiogram. The tricuspid and mitral valves open almost instantaneously, but a period of about $\frac{1}{20}$ second is required before the fall of pressure is transmitted to the jugular vein and the pressure begins to fall (*y* collapse). The inrush of blood into the ventricles

FIG. 107.—Forces supposed to be at work in the production of the third heart sound. Diastolic closure of the auriculoventricular valves. Dotted lines indicate the direction of flow. Black arrows indicate the recoil waves tending to push the cusps together.

[1] The assumption of such a slapping together of the auriculoventricular valves at the end of ventricular filling is not at all incompatible with the fact that a small separation (1–3 mm.) may reappear between them in the latter part of diastole, when the accumulation of blood in the auricles has become sufficient to just force the cusps apart (page 371).

rapidly distends the latter until they reach their full distention, at which the inflow ceases and the cusps of both mitral and tricuspid valves slap together (closing slap in diastole). The end of this inflow may be accompanied by a slight recoil or similar movement of the ventricle, giving rise to the small wave and shock noted at this moment. The intensity of this recoil is probably dependent to a great extent upon the elasticity (elastic tissue) of the ventricular walls; hence its absence in old persons. Whether the feeble third heart sound is due to the slapping together of the valves or is due to some other cause cannot be stated with certainty. After the period of diastasis (slower inflow) has set in, the blood begins to accumulate in the veins, which are distended at first rapidly and later in diastole more gradually.[1] The angle made by these two portions of the venous curve forms the *h* wave. The foot of the *p* wave commences at the end of the period of rapid ventricular filling and corresponds to the crest of the protodiastolic wave upon the cardiogram.

A priori, according to this explanation a protodiastolic sound should be heard in slow hearts because in them the ventricular walls are distended to their full extent early in diastole; in cases of aortic insufficiency because of the high intraventricular pressure which tends to slap the cusps of the valves together early in diastole; in mitral stenosis owing to the peculiar events in the filling of the ventricle (vide page 9), and perhaps in cases in which there is a large amount of residual blood in the ventricle (dilatation) which tends to diminish and shorten the period of inflow. These represent the chief conditions in which it is actually heard. Thayer states that it can be heard at the apex in about 30 per cent. of normal individuals lying upon the left side.

By decades its frequency was as follows; First decade heard in 58.9 per cent.; second decade 84.4 per cent.; third decade 50.9 per cent.; fourth decade 42.3 per cent.; fifth decade 14 per cent.; sixth decade and after 0. It seems to occur in practically every condition, especially in cases with slow hearts, and seems to bear no definite relation to cardiac weakness.

MURMURS.

MECHANICAL FACTORS IN THE PRODUCTION OF MURMURS.

As has been seen above (page 92), when a narrowing occurs in the lumen of an elastic-walled tube through which liquid is flowing, eddies are formed which set the walls of the tube into vibration and give rise to a palpable thrill. Accompanying the thrill a blowing sound known as a "murmur" may be heard over the tube; which, like the thrill, is heard much better below the obstruction than above it, and is transmitted in the direction of the flow. The character of a murmur depends upon the width of the orifice at which it is produced, upon the nature of the walls of the orifice, upon the velocity and tension under which the fluid passes through it, and upon the direction in which the flow occurs.

In this way a valvular orifice may be compared to the larynx with its vocal cords. When the cords are lax and wide apart, the air moving over them in even forced respiration gives no sound; when the cords are approximated a little but still held loosely, it gives a whispered "ch" sound, and when they are held very tense true vocal sound is heard. Similarly, no sound can be heard over the excised heart when the fluid regurgitates through an absolutely patent mitral orifice (Fig. 108); if one of the chordæ tendineæ be stretched and the regurgitation takes place through a small slit whose walls

[1] It is not improbable that, though the cusps are in apposition along the greater part of their line of closure, they are separated at a few points during diastasis.

are flabby (relative insufficiency Fig. 109), a soft low blowing murmur will be heard (the smaller this orifice the higher pitched and more distinct the murmur), while if some more or less hard irregular body, like calcified vegetation, is situated at the orifice, this acts more or less as a resonator, increases the sound and may even give it a roaring or a squeaking (musical) character.[1]

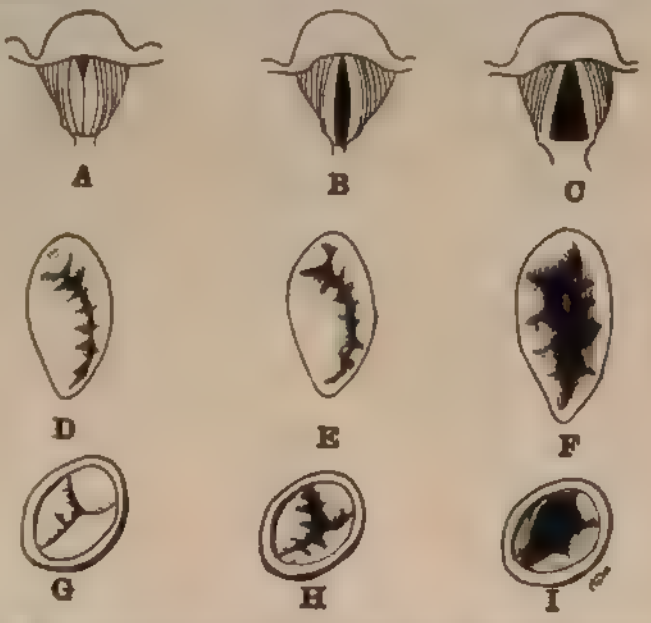

FIG. 108.—Similarity between production of voice sounds and the production of murmurs. (Kindness of the J. Am. M. Asso.) A, B, C, vocal cords. D, E, F, auriculoventricular valves. G, H, I, aortic and pulmonic valves. A, high note; D, G, small leaks producing high pitched murmurs; B, low note; E, H, larger leaks producing low pitched murmurs; C, F, I, very large leaks, producing no murmurs.

Occasionally murmurs become so loud as to be heard several feet away from the chest or even across the room. Such murmurs are usually systolic in time and are often due to calcified vegetations, arterial plaques, or aortic or mitral stenosis. As in the larynx, the character of the sound produced at a valvular orifice is due not only to the size and shape of the orifice, but also to the tenseness of the walls and velocity of blood flow through it, and hence is largely dependent upon the height of the blood-pressure. All these factors, both the widening of the leak and the decreased force of the beat, explain the fact that as the heart weakens under the influence of the lesion the murmur may acutally disappear.

CHARACTER OF MURMURS.

Murmurs may be roughly divided into the following classes: (1) Direct murmurs best transmitted in the direction of the blood flow, as from stenoses or calcified plaques. 2. Regurgitant murmurs due to a flow in the direction opposite to the

[1] Musical or squeaking murmurs are sometimes due to the presence of tense moderator bands stretching across the ventricular cavity and resounding like banjo strings, although usually these bands do not cause murmurs at all.

Very frequently they arise in dilated right ventricles in association with functional tricuspid and perhaps functional pulmonary insufficiencies. They are usually systolic, but sometimes diastolic in time. They are often cardiopulmonary.

usual blood flow (as in mitral and aortic insufficiencies); (3) To-and-fro "machinery" murmurs which occur in both systole and diastole in congenital heart lesions. (4) Rumbling murmurs.

Of these 1, 2, and 3 are more or less blowing or roaring in character, while the rumbling murmurs are devoid of this character, and are rumbling or echoing, more like a series of heart sounds which vary in intensity (mitral stenosis, Flint murmur) than like murmurs due to the passing of a stream through an orifice.

Brockbank claims that these may be produced upon a model by means of a stream flowing through a conical valve from apex to the base of the cone. The mechanism of the production of such murmurs is still very obscure, and further researches are necessary before satisfactory elucidation can be given.

"ACCIDENTAL," "HÆMIC," AND "CARDIOPULMONARY" MURMURS.

Murmurs over the heart without the presence of valvular lesions are so common that autopsy evidence led Laennec to the erroneous belief that murmurs (bruits de soufflet) were of no diagnostic importance whatever.

Such murmurs are designated by various terms: "Haemic," on the assumption that they are always due to anæmia, hydræmia, or other changes in the quality of the blood; "Functional" or "inorganic," because they are not associated with organic lesion; "Cardiopulmonary" or "cardiorespiratory," on the assumption that they arise in the lung above the heart and not in the heart itself; and "Accidental," since they are not associated with any discernible alteration in form or function. These terms are not mutually exclusive; but, since the term "functional" has been used to designate conditions in which there is actual leakage owing to muscular weakness, and since "inorganic" should include both "functional" and "accidental," the term "accidental" appears to be the one most generally useful. Thus, one murmur may be said to be an accidental murmur of hæmic origin, while in another case the accidental murmur may be of cardiopulmonary origin.

Occurrence of Accidental Murmurs.—Potain, who has made the most extensive investigations upon the subject, found such murmurs in one-eighth of all the patients seen in his hospital service. It was present in almost all his cases of Basedow's disease (exophthalmic goitre). In chlorosis the frequency was 50 per cent.; in rheumatism, measles and scarlet fever 20–25 per cent.; in typhoid 10 per cent.; in pulmonary affections, 5–10 per cent. These murmurs were common in subjects in the first three decades of life, reaching maximum frequency at the ages from 20 to 30, and gradually decreased in frequency after the age of 30. For description of the murmurs Potain divided the precordium into the following regions: 1. About the apex (apical zone); 2. Above the apex (supra-apical); 3. Lateral from the apex (para-apical); 4. In front of the infundibulum and conus arteriosus of the pulmonary artery (pre-infundibular); 5. A zone between the pre-infundibular region and the apex (left preventricular); 6. An area behind the sternum (sternal region); 7. A region behind the xiphoid (xiphoid region). The murmurs are most common in the region lying between the pulmonary area and the apex (Potain's left ventricular region), —that is, in the region above the right ventricle and the interventricular septum.

Character of Accidental Murmurs.—These murmurs usually are soft and blowing, and often seem rather superficial. They vary greatly when the patient changes his position. Sometimes they are best heard when the patient is

lying down and diminish or disappear entirely when he stands or sits up; sometimes they appear only when the patient's position is vertical and disappear on his lying down. They also vary with the phases of respiration.

Time of Accidental Murmurs. — As regards their occurrence in the cardiac cycle, accidental murmurs are most commonly systolic in time, though occasionally diastolic. Potain calls attention to the fact that murmurs may occupy either the whole of systole (holosystolic) or only a portion of it. The latter may occur only at the very beginning of systole (protosystolic), so that they accompany or replace the first heart sound. Or, they may be heard in midsystole (mesosystolic), in which case they follow the first sound but are separated from the second sound by the short pause, which is then somewhat shorter than usual. Or, they may occur at the very end of systole (telesystolic) and end, without interruption, in the second sound. According to Potain, the murmurs of mitral and tricuspid insufficiency are heard throughout the entire duration of systole, a view which is confirmed by the graphic records of Einthoven and Weiss and Joachim. The accidental murmurs, however, are confined to only a portion of systole. Potain believes that, as a rule, they are entirely mesosystolic; while Weiss and Joachim, from both auscultatory and graphic evidence (Fig. 110), believe that they also accompany and modify the first sound though they do not replace it; in other words, that they occupy both the protosystolic and the mesosystolic portions of the systole.

Sahli states that accidental murmurs never occupy the very end of systole (telesystolic, Potain, prediastolic), but Potain has shown that though such murmurs are rare they occur occasionally.

Accidental diastolic murmurs are also rather common, and may occur either in the aortic region, behind the sternum, or along the upper left border of cardiac dulness. Occasionally they are heard at the apex. They are usually short superficial puffs following a well-marked second sound and lasting during only a short portion of early diastole.

Differential Diagnosis of Accidental Murmurs. Potain gives the following points in which other murmurs differ from the cardiopulmonary:

1. Pulmonary Stenosis: loud, rough holosystolic murmur, maximum in second left interspace, transmitted toward left clavicle; always accompanied by a thrill. The accidental murmur is soft, often mesosystolic, devoid of thrill.

2. Pulmonary Insufficiency: diastolic murmur maximum in second left interspace, pulmonic second sound absent or diminished. The accidental diastolic murmurs very rarely have their maximum in the second left interspace.

3. Aortic Stenosis: rough holosystolic murmur, maximum in second right interspace, propagated toward right clavicle; accompanied by thrill. The heart is hypertrophied. The accidental or cardiopulmonary murmur in this region is more superficial, soft, and changes in character, change of position.

4. Anæmia: murmur very similar to that of aortic stenosis, but the thrill is less marked and the heart is small or dilated rather than hypertrophied.

5. Aortic Insufficiency: murmur commences exactly at the beginning of the second sound and almost entirely fills diastole, whereas the early pulmonary diastolic murmur follows the second sound often after a short intervening pause, i.e. the murmur is mesodiastolic. Both aortic and accidental murmurs are of wide distribution, embracing the entire precordium, and varying greatly with change of position.

6. Patent Septum of the Ventricles: holosystolic murmur loudest at the third left interspace; rough, always accompanied by a thrill; whereas the accidental and cardiopulmonary murmurs are not

7. Mitral Insufficiency: murmur holosystolic, usually rather rough, maximum at the apex. The cardiopulmonary murmur may have its maximum two or three centimetres lateralwards from the apex, and this is usually associated with a systolic retraction at the apex.

8. Tricuspid Insufficiency: murmur maximum over sternum and xiphoid process. There is an increased area of flatness (hypertrophy of ventricles). This murmur is also increased by leaning forwards so as to throw the heart against the chest wall.

Nature and Causation of Accidental Murmurs.—The facts mentioned above apply to a large number of cases in which murmurs have been heard during life, but in which no leaks and no lesions of the heart were demonstrable at autopsy.

A large variety of factors have been mentioned to explain these accidental murmurs:

Hæmic Murmurs.—Bouillaud was the first to call attention to the fact that murmurs were more readily produced in the less viscous blood of anæmia than under normal conditions; a fact which was subsequently verified by Cohnheim, but Bouillaud himself realized that though anæmia might give rise to some of the accidental murmurs there were many cases in which it could not be a factor. The blood counts made in later decades have entirely substantiated Bouillaud's conservatism. However, numerous observers from Bouillaud's time to the present have adhered to the "hæmic" origin of the accidental murmurs. Sahli goes so far as to state that they may in reality be only venous hums transmitted to the ventricles, though he does not explain why they should be systolic in time. Even though this explanation is inadequate, it is certain that in cases of grave anæmia such transmitted murmurs do arise. They are heard very loudly over the aorta and second right interspace, but are loud, rough, and superficial, quite different from the gentle blow of the usual accidental murmurs.

Functional Insufficiency of the Auriculoventricular Valves, especially of the mitral, was supposed by Naunyn to be the chief cause of the accidental murmur in the pulmonary area. Naunyn believed that this murmur was transmitted from the left auricle directly to the pulmonary artery and thence to the chest wall in the pulmonary area. However, in these cases the murmur may not be heard at all in those areas in which the definite mitral and tricuspid murmurs are best heard. Functional insufficiency of the tricuspid valve has also been assumed, but this is rendered improbable by the fact that these murmurs have a very different distribution from those of the tricuspid and are rarely heard over the xiphoid process. In dogs the writer has found accidental murmurs very common, but, in contrast to the murmurs in tricuspid or mitral insufficiency, these accidental murmurs cannot be heard over the right or left auricle. In man also they are not heard over the region of the right auricle even when the patient is made to lean forward and the walls of that chamber are thus pressed against the chest wall.

Functional Stenosis of the Pulmonary Artery and Infundibulum has been assumed by Luethje in order to explain the production of systolic murmurs in the pulmonary area. It is true that the pulmonary artery makes a sharp bend just behind the second left interspace; and also, as Romberg and others have shown, that often the accidental murmur is increased by pressure with the stethoscope. Against this view are the softness of the murmur, the absence of a thrill, and the fact that it is not transmitted toward the left shoulder, but is well heard over the right ventricle. Moreover, in dogs the accidental murmur may persist in practically every position in which the heart may be held.

Eddy Currents within the Ventricles.—Hilton Fagge has called attention to the fact that eddy currents may arise within the ventricles as the blood passes between the papillary muscles and the trabeculæ carneæ, that these may strike against the bases of the papillary muscles and the chordæ tendineæ, set them into vibration while they are tense during systole, and thus give rise to a systolic murmur. Although Fagge himself believed that this would apply only to a dilated heart, it seems also applicable to a small heart, since during systole the apertures between the walls and the papillary muscles are smaller and more slit like. However, the explanation lacks confirmation.

Similar to this view is the old-time assumption that accidental blowing as well as musical murmurs indicated the presence of a moderator band across the chamber of the right ventricle, but this is not borne out by autopsy experience.

Cardiopulmonary Factors. Laennec in 1826 wrote "In certain persons the pleurae and the anterior borders of the lungs extend in front of the heart and cover it almost entirely. If one examines such a person when his heart is beating more forcibly than usual, the diastole of the heart, compressing these portions of the lungs and forcing the air out of them, alters the breath sounds in such a way that it imitates a blowing murmur or the sound of wood file. But with a little skill it becomes easy to distinguish this sound from a cardiac murmur. It is more superficial, one hears the normal heart sounds below it, and it disappears almost entirely when the patient is made to hold his breath for a few moments."[1]

Physiological experiments have borne out Laennec's claim that the lung moves to and fro with each cardiac cycle (Buisson, Voit, van der Heul, Landois, Meltzer) but have demonstrated that the most sudden movement of the air accompanies the rarefaction of the air within the lung during systole rather than its extrusion during diastole.

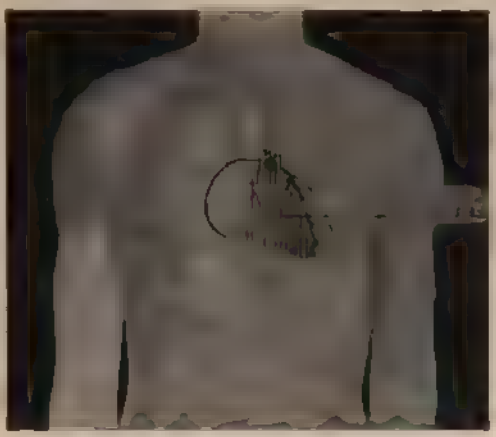

Fig. 109.—Distribution of the accidental murmur.

The cardiopulmonary murmurs formed the subject of an exhaustive study from 1865 to 1894 by Potain, many of whose data have been given above. Potain controlled the findings by auscultation with carefully made cardiograms and experimental studies and found that

1 The cardiopulmonary murmurs are loudest and most frequent in those regions (infundibulum and vicinity of the pulmonary artery) where the movement of the heart is greatest.

2 They occur in regions and in phases of the cardiac cycle at which the cardiogram shows retractions of the interspace (areas of negative pressure with sudden expansion of the lung).

Hence, the systolic murmur is most common over the infundibulum and right ventricle, over which there is usually a systolic retraction (see page 91 and Fig. 89).

If the retraction (fall in the cardiogram) occurs in the middle of systole the murmur is found to be mesosystolic; if at the end of systole the murmur is telesystolic, if the fall is in diastole, the murmur is diastolic. Indeed Potain encountered several cases in which the form of the cardiogram changed upon alteration of the position of the patient, and corresponding to the period of greatest retraction the murmur over the area changed from mesosystolic to diastolic.

This is a surprising confirmation of the theory of cardiopulmonary murmurs. There can indeed be no doubt that cardiopulmonary murmurs are frequent and that they form a very considerable proportion of "accidental" murmurs. Besides the blowing murmurs referred to above, it is probable that many of the so-called "musical" or "squeaking" murmurs are of cardiopulmonary origin and are really piping râles produced by the

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[1] "Chez quelques sujets les plèvres et les bords antérieurs des poumons se prolongent au-devant du cœur et le recouvrent presque entièrement. Si l'on explore un pareil sujet au moment où il éprouve des battements du cœur un peu énergiques, la diastole du cœur comprimant ces portions du poumon et en exprimant l'air altère le bruit de la respiration de manière à ce qu'il imite plus ou moins bien celui d'un soufflet donné par le cœur lui-même; il est plus superficiel, on entend au dessous le bruit naturel du cœur, et en recommandant au malade de retenir pendant quelques instants sa respiration, il diminue beaucoup ou cesse presque entièrement."

to-and-fro movement in the lung during either phase of the cardiac cycle. Other *râles* of *cardiopulmonary origin* more closely resembling the sonorous and crepitant râles of respiration are also very common along the margin of the left lung. Moreover the breath sounds themselves are frequently modified by the cardiac movements, giving rise to the so-called *cog-wheel type of breathing*, in which inspiration is interrupted by a series of small clicks and jerks coincident with and due to the effects of cardiac contractions upon the air in the lungs. The cog-wheel type of breathing is often associated with slight changes in the overlying lung and is thus often a premonitory sign of pulmonary tuberculosis.

Differentiation between Cardiopulmonary and other Accidental Murmurs.—However, in spite of the frequency of cardiopulmonary murmurs, it is probable that Potain erred in ascribing all accidental or non-valvular murmurs to this origin. In the first place, many such murmurs are audible over the area of cardiac flatness several centimetres from the lung borders, when breath sounds which are of equal loudness over the lung cannot be

FIG. 110.—Graphic record of an accidental murmur. (After Weiss and Jonckheim.)

heard at all at these sites. Secondly, the murmurs can be well heard directly over the exposed dogs' hearts when the lung has been entirely retracted, and when valvular insufficiencies and stenoses can be absolutely excluded.

For the present, therefore, it must be admitted that there are still many uncertainties in the differentiation between cardiopulmonary and other accidental murmurs. *The diagnosis of the former must be confined to murmurs of distinctly superficial quality which are heard loudest over the lung borders and are absent or much diminished over the area of cardiac flatness*, and which vary with change of position. The diagnosis may be considered as rendered probable if the area over which the murmur is heard moves toward the sternum in inspiration and away from it in expiration, corresponding to the movement of the marginal strip of lung. If the reverse is the case and the area of intensity extends lateralward in expiration and recedes toward the sternum in inspiration, the murmur is more likely to arise within the heart.

Imitations of the Heart Sounds. A remarkably accurate method for imitating the heart sounds, reduplications, and rough or blowing murmurs has been used for the past three years by the writer's colleague Dr. Charles W. Larned. This is carried out by placing the palm of the observer's hand tightly over his ear, and then tapping upon the elbow with the finger tips of the other hand. The blow must be struck with loose finger-joints. Its force can be varied to suit variations in the loudness of the sound. Dull and distant sounds may be imitated by light blows of the finger or by raising the palm of the hand from the ear, snapping sounds by pressing the hand tightly upon the ear and executing a sharp stroke. Blowing murmurs are reproduced by a gentle stroking of the

elbow. Dr Henry Lee Smith has modified this procedure by striking the blows directly upon the back of the hand, instead of the elbow, a method by which sharper and more snapping sounds can be produced. He is able to give a very accurate reproduction of the presystolic rumble and snapping first sound of mitral stenosis by bringing all the four fingers down upon the knuckles or metacarpals in as rapid succession as possible, a manœuvre which is best executed by a quick pronation from the elbow. The blow struck with the index finger (snapping first sound) should be somewhat louder than the rest.

While these methods are excellent for demonstrating to one student at a time, they cannot be used for demonstrating to a whole group simultaneously. For this purpose the writer has resorted to the somewhat cruder method of executing the same taps and strokes upon the top of a derby or even a soft felt hat. This imitation is not quite so accurate, and the snapping and rumbling quality are not reproduced, but nevertheless it enables the instructor to point out the salient features to all and to illustrate their main variations and relations to the events of the cardiac cycle.

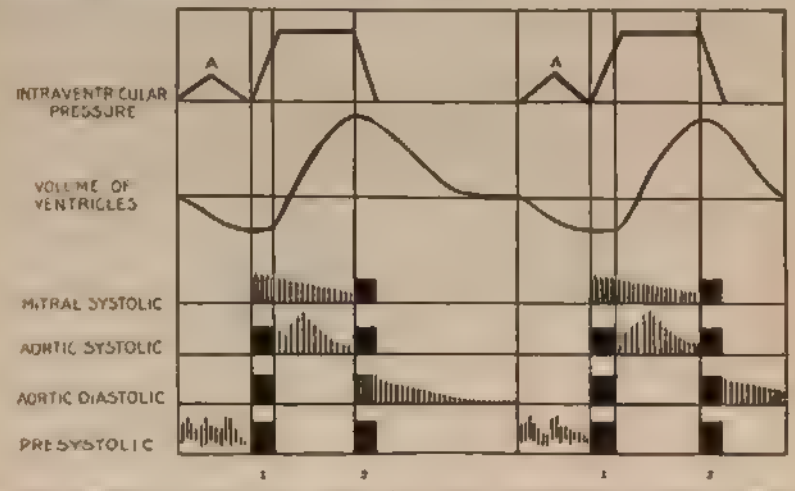

Fig. 111.—Diagram showing the relation of the more common simple murmurs to events of the cardiac cycle. Solid black bars indicate the heart sounds. Vertical parallel lines reaching to the base indicate blowing or rough murmur. Wavy vertical lines not reaching to the base indicate a rumble.

The exact method for the reproduction of each sound or murmur can thus be indicated schematically by designating the finger to be used (I = index, M = middle, R = ring finger, L = little finger) and the accent of the sound '. Time intervals may be shown by dashes, and rapid succession of the split sounds by bracketing the corresponding letters. Murmurs are indicated by stroke.

Thus I' — I = Normal first sound at the apex, I — I' = Normal first sound at aorta, stroke I' = Mitral murmur, I — (I'M) = Split second sound, (IM — I) = Split first sound, (I M' — I) = Presystolic gallop, I — I'-M = Protodiastolic gallop, I stroke I = Mesosystolic murmur, LRMI' — I = Presystolic murmur, I — stroke = Diastolic blowing murmur replacing second sound, I — I stroke = Diastolic murmur following the second sound, LRMI' stroke I = Presystolic systolic murmur of mitral stenosis, Gentle to-and-fro rubbing of skin = Pericardial friction.

Relations of the Simple Murmurs to Events of the Cardiac Cycle. The relations of the simple cardiac murmurs to the contractions of the cardiac chambers, as well as to the filling and emptying of the ventricles, is shown in Fig. 111. The mechanism of their production will be discussed in detail in connection with the valvular lesions to which they correspond. It will be seen, however, that the mitral systolic murmur begins coincidently with the first heart sound before the blood flows into the aorta, and that it continues throughout systole, that the aortic systolic murmur follows the first sound and is loudest in midsystole, that the aortic diastolic murmur is loudest in early diastole, when the filling of the heart and the regurgitation are most rapid, and that the presystolic rumble is produced by the inrush of blood into the ventricles during auricular systole.

SINGLE MURMURS.

Time.	Character.	Phonetic[1] equivalents.	Distribution.	Clinical condition.
Presystolic	Rumbling, occasionally blowing	ftat-ta; trat-at; tr-r-rub-dub	Apex only, lower precordium between parasternal line and sternum	Mitral stenosis; tricuspid stenosis.
Systolic	Blowing or roaring. Enters into or replaces as well as follows first sound. Uniform or decrescendo	shush-dub; jjje-dub, esh-dub; faf-tam	Over body of heart, at apex and to axilla, often at back. Over lower sternum and neighboring precordium	Mitral insufficiency; tricuspid insufficiency.
	Blowing or roaring; *follows* first sound; has a crescendo character in mid-systole and decrescendo in late systole	lussch-dub; taf-dub	Loudest over 2d right interspace; thrill also in vessels of neck. Not so loud at apex	Sclerosis of aorta; aortic stenosis, congenital heart lesion.
	Similar in character to aortic systolic murmur	lussch-dub; taf-dub; taf-tam	2d left interspace and to left of sternum (thrill). Elsewhere over chest (thrill)	Pulmonary stenosis, congenital heart lesion, aneurism.
Mesosystolic or telesystolic (prediastolic)	Soft blowing, uniform or decrescendo	lupff-dub; taf-tat; luff-dub	Over entire precordium, esp. 2d and 3d left interspace. Varying with change of position. Not transmitted beyond apex	Functional, accidental, or anæmic murmur. Anæmic fever; neurasthenia etc.—sometimes organic (?).
Diastolic	Blowing	lupd-shah; tam-taf; lup-dush; lup-shah	At 2d rib near sternal margin; loudest over sternum at level of 2d left interspace and in the latter near the sternal margin	Aortic insufficiency.
			At 2d left interspace and right sternal margin; also to right of sternum	Pulmonary insufficiency.
			At 2d left interspace and sternal margin	With no other marked signs of valvular insufficiency. Abnormal murmur (Potain, Graham, Steele).
Mid-diastolic	Rumble	lub-dub-tra.	At apex only	Mitral stenosis; some cases with pericardial adhesions, etc.

[1] These phonetic equivalents most closely imitate the cardiac sounds when the consonants are prolonged as much as possible.

COMBINED MURMURS.

Time.	Character.	Phonetic equivalents.	Distribution.	Clinical condition.
Presystolic (Flint murmur), systolic, and diastolic	Presystolic rumble, systolic blow; diastolic blow	ftaftash; tr-r-rub-dush	Blow loudest at 2d right and 2d left interspaces; at apex and out in axilla. Rumble over apex only	Vegetation at aortic valve, aortic insufficiency. Sometimes, but not necessarily, mitral insufficiency, occasionally also mitral stenosis.

COMBINED MURMURS (Continued).

Time.	Character.	Phonetic equivalents.	Distribution.	Clinical condition.
Systolic and diastolic	Systolic and diastolic blow	taf-tash; lush-dush; shush-shush	2d right and left interspace, sternum, left sternal margin transmitted to arteries	Aortic insufficiency with aortitis; aortic stenosis.
Same . . .	Same...	Same . ..	Loudest at left sternal margin, thrill maximum to first and second left interspace	Pulmonary stenosis and insufficiency; open ductus arteriosus, other congenital heart lesions.
Irregularly in both systole and diastole	Soft, superficial, scratchy	slush-dush, slush-dush-da	Over the entire precordium, especially over the area of absolute dulness; increased by pressure with stethoscope	Fibrinous pericarditis.
Accompanies both heart sounds and both breath sounds	Pleuropericardial.	.. ..	Over relative cardiac dulness only; scratch simultaneous with respiration as well as cardiac cycle. Increased by pressure with stethoscope	Pleuropericarditis.
	Crepitant; small explosive rales	.. .	Over relative cardiac dulness only	Emphysema. Interstitial emphysema.

VASCULAR SOUNDS AND MURMURS.

Arterial.—Besides the murmurs transmitted from the heart, murmurs also occasionally arise in the arteries themselves. A systolic murmur and an audible first sound (pistol-shot tone) may be produced by pressure with the stethoscope over the arteries, but without exerting a definite pressure it may often be found accompanying the dilatation of markedly pulsating arteries, as in aortic insufficiency and with dicrotic pulses, etc. The eddies arising in an aneurism usually give rise to a rough or blowing systolic murmur which may be transmitted for a considerable distance along the arteries. In aortic insufficiency a double murmur (sytolic and diastolic) may be heard over the arteries (Duroziez).

Venous.—A sound is heard over the jugular vein, especially over the jugular bulb just above the clavicle, in cases of marked anæmia, chlorosis, etc. (Camac). The murmur is humming or roaring in character and occurs during both systole and diastole (humming-top murmur, "bruit du diable," etc.). Weiss and Joachim have registered the sound and have shown that it never ceases. As shown by Cohnheim the anæmic blood flows more rapidly than does normal blood, probably owing to its lower viscosity; and both these factors facilitate the production of a murmur. However, it has not yet been shown that the murmur is loudest at those periods of the cardiac cycle during which the flow in the veins is most rapid.

MURMURS AS AN AID TO DIAGNOSIS.

It is evident from what has gone before, as well as from the consensus of medical practice, that auscultation furnishes a most important means of diagnosis of cardiac lesions. It is equally evident that each abnormal sound may be associated with any one of several clinical conditions, which must be still further differentiated from one another, not only by the murmur but by its distribution, transmission, and variations, but particularly by the other methods of physical examination, graphic methods, and X-ray examination. The examiner should not content himself with a simple designation of the lesion, but should become fully conversant with the disturbance of function in all parts of the circulatory system, and with its remote secondary effects.

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PART II.

I.

PRIMARY CARDIAC OVERSTRAIN.

It has long been known that heart failure may arise from simple overstrain of the heart without the intervention of any actual cardiac symptoms. This condition usually remains acute and ends in rapid recovery, but it may also become chronic and reduce the patient to lasting invalidism. In its worst form such a purely functional weakening of the heart may result in death.

This conception was first introduced by Stokes in 1854, and was confirmed later by studies of Clifford Allbutt, A. R. B. Myers, and Peacock in England, and da Costa in America. Their articles were collected, translated into German, and published, along with an excellent monograph upon the subject, by Johannes Seitz, of Zürich, in 1875, bringing them to the cognizance of the German writers. In 1886 v. Leyden added important contributions. In 1898 the matter was subjected to clinical experiment by Theodor Schott, whose conclusions have been disputed by a host of later and more careful observers.

The most interesting, extensive, and complete of all these papers are those of da Costa, based upon several hundred cases occurring among Union soldiers of the Civil War. It is impossible to do justice to these studies in a brief abstract. His presentation is so complete and so nearly a model of clinical study for its time that the reader is urged to consult the original publication.

CLINICAL CASES.

A very typical case of da Costa's series is illustrated by the following history.

Case I.—Irritable heart, chiefly from hard service; recovery. —Wm. Henry H., private 68th Pennsylvania Vol., admitted into the Turner's Lane Hospital in Philadelphia, November 2, 1863, having just returned from a furlough. He enlisted in August, 1862, at the time in good health, though he had suffered occasionally from rheumatism. He did a great deal of hard duty with his regiment. Some time before the battle of Fredericksburg, he had an attack of diarrhoea; after the battle, he was seized with lancinating pains in the cardiac region, so intense that he was obliged to throw himself down upon the ground, with palpitation. These symptoms frequently returned while on the march, were attended with dimness of vision and giddiness, and obliged him often to fall out from his company and ride in the ambulance. Yet he remained with his regiment until July 4, 1863, when he was wounded at the battle of Gettysburg. The wound healed in about one month; but the cardiac symptoms became worse, and violent palpitations ensued upon the slightest exertion, sometimes also whilst in bed, obliging him to rise. There was soreness in the cardiac

region, and a constant dull pain. The impulse was extended slightly jerky, 96, and of irregular rhythm, some beats following one another in rapid succession, the first sound was feeble, the second very distinct. The man did not look sick. Height 5 feet 7 inches, measured 31 inches around the chest one inch below the nipple, he did not smoke, chewed tobacco in moderation.

The patient did not improve under aconite, but under digitalis the impulse became quiet and 78, and on March 24 having previously done duty as orderly, he was detailed on police duty, and his treatment stopped. The heart continuing to act regularly, he returned to his regiment May 3, 1864.

Another case, quoted from v. Leyden, illustrates the various phases of the malady very well.

Case II. Carl Timm, butcher, aged 30. Family history negative. Syphilis ten years before, quiescent for several years. Otherwise always healthy. Performed military service for 5 years without any trouble. In the fall of 1880 became a butcher in the Charité Hospital where he had to lift and chop sides of beef weighing 200 lbs. The first symptoms appeared suddenly on the afternoon of December 30, 1880, during an ordinary day's work, when he felt a severe pressure in the pit of the stomach, preventing him from taking a deep breath and causing him to stop in his work. At this time he noticed palpitation and irregularity of the heart. For several days after this he did no heavy work and then felt well. When he tried to do heavy lifting again the same pain and sensation of pressure returned, and though he continued his work he was compelled to stop for breath from time to time. On Feb. 17, 1881, the pain became very intense and he entered the hospital on Feb. 21.

Physical Examination.—Patient is a very well-built young man, well muscled, well nourished but not fat. Complexion florid but healthy looking. No dyspnœa or cyanosis. Moderate œdema of lower extremities. Patient complains only of palpitation of the heart. The pulse is strikingly irregular so that it is impossible to count. The radial arteries are small, blood-pressure apparently low. The cardiac impulse is intense, vibratory, and very irregular. Apex impulse is in 5th left interspace 2 cm. beyond mammillary line, well marked, readily palpable. Cardiac dulness begins above at 3d rib extending below to 6th rib, and reaching just to the right of the sternum. Heart sounds are feeble and unequal but clear. Lungs clear. Liver and spleen are not enlarged. Urine 500 c.c., sp. gr. 1023, no albumin.

Ordered rest in bed, ice-bag over heart, infusion of digitalis every 2 hours. Within a few days symptoms and œdema had subsided, but the irregular heart action persisted. By March 20 he was well enough to be discharged, with the following note. Apex beat 0.5 cm. to left of mammillary line, first sound at apex loud and ringing, second sound distant but clear, pulse irregular, examination otherwise negative.

This represents the first stage of his illness, in which the following features are noteworthy. 1. A very strong and perfectly healthy young man suffers from heart failure as the immediate result of overstrain. The first attack came on suddenly while at work and passed off soon, but attacks recurred whenever the patient did heavy work, and he was compelled to enter the hospital. 2. Physical findings: heart dilated especially in the longitudinal axis, weak apex beat (dilatation of left ventricle), great cardiac irregularity. 3. Relatively rapid improvement after rest in bed and digitalis. 4. The heart then returned to almost normal size, but the irregularity in rhythm persisted.

Second Stage.—Patient returned to his old work in spite of warning and within two months œdema of the legs had again set in and he was confined to bed for eight weeks more. Returning to work again he could perform only very light labor and very soon returned once more to the hospital for seven weeks with still more marked œdema. Once more these disturbances disappeared after rest and

digitalis but thereafter the slightest work caused palpitation and the feeling as though there were a tight cord about the chest. He also felt pain in the region of the liver. At this point he re-entered the hospital. Pulse 180 small irregular. Face flushed, no cyanosis. Expression depressed. Skin normal, oedema of feet and legs. Gangrene of big toe of right foot. Respiration a little rapid, dyspnoea only on exercise, but while walking he often stops to catch his breath. Occasionally he has attacks of dyspnoea lasting about 10 minutes beginning with a feeling of pressure in the region of the heart. He then feels as though hot liquid were pouring from the heart upwards to each side of the neck.

Physical Signs. Apex beat in 6th left interspace in anterior axillary line, soft and easily compressed. Heart therefore much enlarged, sounds clear and fairly loud; action markedly irregular. Liver enlarged and tender.

Ordered rest in bed, digitalis, morphine at night. Patient became much better within twenty-four hours; pulse then 68 per minute. The attacks of dyspnoea almost disappeared. Urine 1500 sp. gr. 1020. Within six days all cardiac symptoms had disappeared. March 11. Pulse 52. Feels well, no pain. Apex beat in 5th left interspace 3 cm. beyond mammillary line, moderately forceful. Heart sounds clear but irregular. He still occasionally has feeling of pressure in chest.

Features of second stage: 1. Dilatation of heart much more marked than before. 2. Very rapid and very irregular heart action. 3. Definite attacks of pain in heart and feeling of pressure (anginoid in character), with radiating pains in shoulder and arm. 4. Swelling of liver (failure of right heart). 5. Return to almost normal under treatment, diminution in size of left ventricle. 6. Intercurrent affections, small infarct of lung, pressure gangrene of great toe, recovered from.

Third Stage. Returned to the hospital in July 1885 (two years later). He has been able to do very little since last admission. Now much emaciated, face thin, appears depressed. Cheeks and lips slightly cyanotic. Respiration dyspnoeic and stertorous. No orthopnoea. Moderate oedema of shins.

Cardiac impulse seen in 5th to 7th left interspaces, apex beat felt in 7th in axillary line forcible. Heart rate about 132 irregular. Cardiac dulness 19 cm. from left sternal margin. Upper limit of cardiac dulness as before begins at 3d rib. Liver readily palpable. Sounds loud, more or less short but no murmur. Did not remain in hospital, but on October 4, 1885 was brought in again in collapse. Marked cyanosis, extremities cold, oedema of legs up to knees. Heart as before, sounds still clear. Pulse 150. Liver a hand's breadth below costal margin. Ordered digitalis, also camphor subcutaneously, tea with cognac. At midnight collapse more marked, very marked dyspnoea and cyanosis, threw himself to and fro, groaned loudly. Pulse not palpable, not revived by camphor or ether injection. At 2 A.M. became quiet, stertorous breathing set in at 3 A.M., died quietly at 3.15 A.M.

Autopsy. Marked oedema of legs. Both lungs slightly retracted, slightly adherent over apices. Pericardium distended, little fluid. Heart markedly enlarged, more than twice the size of patient's fist, especially in the longitudinal axis. Left ventricle more dilated than right. Distance from insertion of pulmonary artery to apex 13 cm., to right border of heart 10 cm. Length of left ventricle 15 cm. Little epicardial fat. Valves normal, aortic valves close perfectly. Papillary muscles well developed, some trabeculae flattened and undergoing fibrous changes. A fibrous patch is seen on the interior surface of the left ventricle. Endocardium otherwise delicate, showing some yellow areas of fatty degeneration of the endocardium and papillary muscles. Cut surface of heart muscle shows cloudy swelling. Left auricle markedly dilated. Right ventricle appears pale with spots of yellow. Lungs, oedema of bases. Liver markedly enlarged, definite nutmeg liver. Kidneys large, dark red, harder than normal.

Microscopic examination shows extensive fatty degeneration of muscle-fibres but only in the inner layers. No interstitial changes, no changes in blood vessels or nerves of the heart. Here and there the interstitial strands of connective tissue appeared thicker than normal but without cellular infiltration.

ETIOLOGY.

In da Costa's 200 soldiers, well-marked fever preceded the overstrain in 17 per cent.; diarrhœa (among which there may have been many mild cases of typhoid fever) 30.5 per cent.; hard field service, particularly excessive marching, 38.5 per cent.; wounds, injuries, rheumatism, scurvy, ordinary duties of soldier life, and doubtful cases 18 per cent. Contrary to the belief of many observers, tobacco did not seem to be an etiological factor in his series.

Allbutt gives the following etiological factors of cardiac overstrain: gymnastics, rowing, Alpine climbing, long-distance running, intense fits of anger or emotion, sexual excesses. Overstrain is very frequent among miners, metal workers, carriers of heavy burdens, blacksmiths, moulders. Morton Prince calls attention to the development of cardiac dilatation under severe mental strain, as in a civil service examination. Anæmia and chlorosis (Henschen), apparently mild illnesses, intestinal disturbances, acute alcoholism, and febrile diseases (Dietlen) are also frequent causes. Sexual excess is an important factor, especially in men; but its effects are usually more marked in hearts already weakened from other diseases or from valvular lesions than in perfectly healthy hearts.

Myers, Allbutt, and Schott have shown that tight belts, uniforms, and corsets displace the heart upward, embarrass its action, and predispose to overstrain. Indeed Myers found that cavalry soldiers with tight belts suffered more from long rides than infantry from marching the same distance.

SYMPTOMS, SIGNS, AND CLINICAL COURSE.

The chief symptoms are dulness, excitability, nervousness, loss of sleep, loss of appetite, restlessness, buzzing in the ears, vertigo, muscæ volitantes, palpitation of the heart, usually very severe and often associated with a feeling of pressure or constriction over the chest. This may be very distressing, but does not, as a rule, cause the patient to remain absolutely still nor give him the fear of sudden death, though da Costa mentions cases in which the precordial distress was great enough to cause soldiers to fall to the ground in the midst of battle.

Pain over the precordium and the left shoulder, occasionally down the arm, increased on inspiration and on coughing.

Dull headache, dizziness, especially on bending over, sleeplessness, indigestion, tympanites, and diarrhœa are common

The patient often wears an anxious expression and there are usually pallor and more or less cyanosis. Pulse is usually small, feeble, rapid, and often irregular. The cardiac impulse may be barely or not at all visible, but on percussion the area of relative cardiac dulness is usually found to be enlarged considerably to the left both downward and upward, and often also to the right as well This corresponds to the dilatation of the left ventricle and of both auricles (*i.e.*, diameters MR and ML, Fig. 84, are much increased).

On the other hand Katzenstein has shown that in just these cases the impulse may be exceptionally strong and impart a heaving to the whole chest, even though the heart be much dilated, failing, and devoid of the slightest trace of hypertrophy A systolic retraction is usually seen over the greater

part of the precordium of these overworking hearts (Fig. 89), corresponding to the contraction of the right ventricle (page 91). Occasionally in rapid and irregular hearts this appearance is somewhat puzzling and has led some clinicians to dictate notes of "delirium cordis" where this condition was not present at all.[1]

The area of relative cardiac dulness is much enlarged (Fig. 112), especially to the left, both downwards, corresponding to the dilatation of the ventricle, and upwards, corresponding to the auricle. In more severe cases, especially with marked cyanosis, the dulness is enlarged also to the right from dilatation of the right auricle. Occasionally this dilatation may have passed off before the patient has been seen by the physician and only the other symptoms and signs persist, but it is safe to assume that it has been present at an earlier stage of the disease.

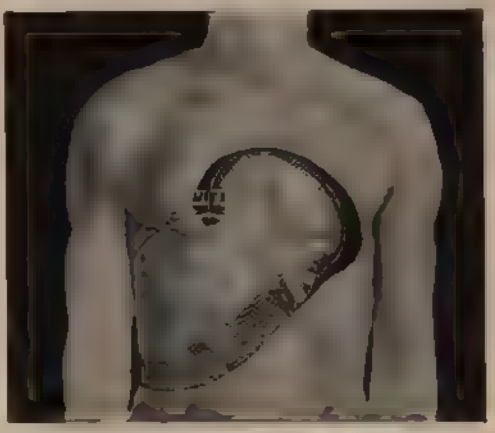

Fig. 112. Cardiac dulness in a Layman's case upon his three successive admissions. I. II. III.

The heart sounds may be either very distant and feeble or very short and sharp, corresponding to the two types of cardiac impulse. They are usually unaccompanied by murmurs, but in an irregular heart may be of uneven intensity. The second pulmonic is usually the loudest sound heard. The clearness of the first sound is often altered by a reduplication, especially in rapid hearts, or by the presence of a soft blowing systolic murmur, which is usually loudest over the pulmonic or tricuspid area, but occasionally also heard to the anterior axilla. These sounds do not always but may sometimes correspond to the presence of functional insufficiency of the mitral valve (vide page 323), in other cases to anæmia. It is, however, extremely difficult or sometimes impossible to decide absolutely whether such an insufficiency is present.

The pulse is usually rapid, ranging from 80 to 160 per minute, small, and weak, in many cases irregular in both force and rhythm. In less severe cases there are only occasional extrasystoles (Schott), in the more advanced there is an absolutely irregular rhythm which persists even after the rate slows. There is often persistent tachycardia without dyspnœa, lasting for even weeks or months.

Clinical Course.—In some cases, however, all the signs and symptoms of overstrain may be present without any irregularity whatever, but often associated with a rapid and regular pulse. Occasionally the pulse may be regular only while it is rapid, but becomes irregular as the rate diminishes. In many cases no murmurs or other signs of valvular insufficiency are

[1] The term delirium cordis is used rather indefinitely to designate conditions varying between extreme irregularity with tachycardia and true fibrillation of the heart. The onset of the latter is, however, not consistent with the existence of life.

encountered, while in still others a relative or functional insufficiency of the mitral or tricuspid valve results from the cardiac dilatation, with some embarrassment of the heart resulting therefrom in addition to the original failure. Systolic (functional) murmurs are heard in these areas, and the stasis is still further increased by the regurgitation of blood. The œdema becomes extreme, hydrothorax may set in, and death soon results. As in the case of da Costa's patient under discussion, the progress may be stayed somewhat by occasional treatment and rest. If the latter is sufficient and the disease not too far advanced, the patient's life may be saved.

The liver, as in Case II, enlarges when the condition becomes severe and tricuspid insufficiency has set in. Its edge is then smooth and varies in consistency from being rounded and so soft as to be palpable only with the side of the index finger to almost board-like hardness. It is always smooth. In severe cases jaundice may be present and the liver may pulsate.

The abdomen is often distended with gas, a factor which contributes largely to the cardiac discomfort by pushing up the diaphragm. In the later stages of heart failure ascites may be present.

The genitalia show œdema only in the later stages of the disease.

The lower extremities are often œdematous, the swelling first manifesting itself about ankles and shins.

The urine during the period of heart failure is usually scant, less than 900 c.c. (30 ounces) for 24 hours,—owing to diminished rapidity of blood flow. It is then of high specific gravity (1020 and over), and often contains albumin and casts. In extreme stasis numerous epithelial, coarsely and finely granular, and hyaline casts are seen in every field of the microscope.

Blood. The blood picture may vary from a moderate anæmia to a real polycythæmia, dependent upon the condition of the patient before the over-exertion.

The sputum may be scanty and mucous, or profuse, frothy, and albuminous, dependent upon the relative strength of the right and left ventricles. In rare cases hæmoptysis results during the exertion from engorgement of the pulmonary capillaries.

Transitory Cardiac Dilatation. A particularly instructive series of cases studied with modern methods are those reported by Hornung (1908). Among 1100 cases which he watched with the X-ray during the past seven years he has met with a number who usually showed perfectly normal hearts but were subject to acute dilatation after overstrain. This was particularly frequent in persons who had used alcohol to excess, in those who had recently suffered from infectious diseases, and in anæmic individuals. The attacks of dilatation are brought on by fright, high altitudes, excitement, over-exertion, etc. Sexual excitement might be added to this list. Hornung returns to the old view of Seitz, Allbutt, and v. Leyden, that cardiac overstrain with acute dilatation is much more common than might be supposed from the work of Moritz and his pupils.

For a long period, however, he may be expected to be more subject to other attacks than before, although by care he may remain free from them. Just how long this susceptibility may last varies with each case,

but da Costa has shown us that after carefully sparing the patient from all severe effort for weeks or even months, he may again perform even such severe efforts as are entailed on cavalry charges and forced marches without injury and may lead a life of perfect health.

The other side of the picture is shown by v. Leyden's case. This man returned to work in spite of the discomfort. The latter became worse, and after bearing it for three months he entered the hospital with a heart already dilated and permanently irregular, and with well-marked œdema of the limbs. Definite heart failure had set in. From this he recovered under rest and treatment with digitalis. His heart resumed almost normal size, his œdema disappeared. The circulation once more returned to almost normal, but one permanent injury had been done for which the treatment was of no avail. The heart action had become irregular and remained so.

The commencement of permanent absolute irregularity in rate (pulsus irregularis perpetuus) (see Part I, Chapter IV) at this stage is a very common occurrence in overstrained hearts, and seems to be one of the most important factors in determining the subsequent course of the disease (see page 123). When the irregularity persists it adds its own mechanical effects on the circulation to those already present and increases the overstrain.

When a life of strenuous muscular work is continued by such a patient the result is inevitable. Strain follows strain, and the condition brought about by the first failure is exaggerated with each successive day's work. The attacks of pain and pressure in the thorax (anginoid attacks) increase in severity and frequency. The heart dilates more and becomes correspondingly weaker. Blood stagnates in the veins, first in the more dependent portions, causing œdema of the ankles, shins, thighs, genitalia, then enlargement of the liver and ascites from stasis in the portal system, finally œdema of the face and arms. The heart dilates still more; the mitral and tricuspid orifices no longer close. After each attack he is less vigorous than before, and greater care must be taken to avoid exertion. For the manual laborer such a life may be at once impossible and intolerable, but the littérateur, the scholar, the scientist, and the man of affairs may be saved for years to a life of quiet but none the less useful activity in spite of a considerable degree of cardiac break-down.

DIAGNOSIS.

The diagnosis of primary overstrain of the heart is not always simple. It is always a question not of whether the heart has been overstrained but of whether this weakening is primary, and whether the heart was perfectly healthy before the effort was made. If the heart, muscle, or valves were in any way diseased before the effort, the overstrain may be considered as secondary to that lesion. Accordingly the diagnosis rests upon the previous history, upon the nature, duration, and sequelæ of previous infectious diseases, upon the degree of arteriosclerosis, and upon the general health of the patient before the onset of the trouble.

Latent myocarditis, fatty degeneration, and arteriosclerosis are particularly difficult to exclude. A mild grade of myocarditis may have given no symptom whatever in daily life, but become apparent when exercise is violent. A mild grade of arteriosclerosis is practically universal among

persons past middle age, but if considerable efforts had been made without symptoms of cardiac insufficiency these may be disregarded. When symptoms of heart failure occur suddenly in a robust individual during or after some intense muscular or nervous effort, acute cardiac dilatation and overstrain may usually be diagnosed with certainty, but, like hysteria among the nervous diseases, it should be arrived at only after a process of careful exclusion.

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II.

PATHOLOGICAL PHYSIOLOGY OF EXERCISE, CARDIAC OVERSTRAIN, HEART FAILURE, AND COMPENSATION.

PHYSIOLOGY OF EXERCISE.

It is evident from the foregoing examples that muscular efforts which lead to cardiac overstrain are in themselves merely the exaggeration of ordinary exercises. To understand these effects it is necessary first to understand those of ordinary exercise.

McCurdy has classified exercises as—

1. Exercises of speed, like running, chest weight exercises, etc., in which the individual movements require little effort, but the main effort lies in the rapidity with which they are repeated.
2. Exercises of endurance, as in long-distance running, prolonged walking, forced marches, etc., in which the movements are neither difficult nor especially rapid and the element of strain sets in only with the onset of fatigue.
3. Exercises of strain, as lifting heavy objects, wrestling, etc.

Exercises of Speed.—The cases of cardiac overstrain reported by Allbutt and da Costa represent overstrain from exercises of endurance; those by v. Leyden and Münzinger represent exercises of strain.

Masing, Erlanger and Hooker, Dawson and Eyster, and Gordon have investigated the effect of exercises of speed such as rapid weight-lifting, running, etc., upon man. The three last named have found that in individuals in training, whose circulation is least affected, mild exercise causes either no change or else a fall of blood-pressure. Tangl and Zuntz also found this in horses and a similar period, though of short duration, in dogs running on a tread-mill.

In all muscular work an increased amount of CO_2 is given off from the muscles and acts as a hormone[1] which sets into play the following physiological mechanisms:

1. Vasodilation in the muscles, diverting four or five times as much blood through this channel (Chauveau and Kaufmann).
2. Acceleration of the heart, at first through diminution in the vagus action, and in the later stages of prolonged severe exercise chiefly through stimulation of the accelerators (Hering, Bowen).
3. Vasoconstriction, especially in the splanchnic vessels, which tends to counteract the effect of the vasodilatation in the muscles.
4. Stimulation of the augmentor fibres, and perhaps also of the heart muscle, directly, causing an increased force of contraction (higher maximal pressure) and an increased systolic output (higher pulse-pressure). Stimulation of the augmentor fibres also, as a rule, causes increased cardiac tonicity.

[1] Hormone, a substance generated in one part of the body which circulates in the blood, reaches and sets into activity another organ, thus playing the rôle of a "chemical messenger." (Cf. Starling, E. H.: On the Chemical Correlation of the Functions of the Body, Lancet, Lond., 1905, ii, 391, 423, 501, 579.)

The heart of the trained athlete is habitually throwing out an amount of blood suited, not to the needs of the moment, but to the needs of the periods of exercise to which he has accustomed himself. The systolic output is above normal when the exercise (and hence the increased production of CO_2) is slight. The heart is thus able to take care of the excess CO_2 production in exercise without increasing its output; and hence the vasodilatation in the muscles is the only factor influencing the blood-pressure. When the exercise becomes severe the other mechanisms begin to play a rôle.

In normal but not trained young men Masing found that upon lifting and lowering a weight with the feet the blood-pressure (maximal) and pulse-rate rose at once to a constant height, where they remained until the exercise ceased. They then fell almost immediately to the original level. The

FIG. 113.—Alterations of blood-pressure due to rapid lifting of light weights with the feet. (After Masing, *Deutsches Arch. f. klin. Med.*, vol. lxxiv.) A. Normal young man. B. Man aged 68.

writer has found that the minimal pressure rises also, but less than the maximal, the pulse-pressure being increased. In middle-aged persons Masing found that the pressure rose higher, and on cessation of the exercise required several minutes to reach the original level; while in very old persons the rise was still greater and neither pressure nor rate returned to normal for a considerable period. The response is proportional to the effort.

When exercise is continued in normal young persons and the organism readapts itself to the effort (the "second wind" setting in), blood-pressure and pulse-pressure again fall to a fairly constant level (Dawson and Hatfield). This probably explains why the heart-rate of well-trained Marathon racers is sometimes slow at the finish. In animal experiments it finds its analogy in the improved cardiac action observed as a result of clamping the thoracic aorta, and represents the response of the heart to a strain which is not excessive.

The weaker the individual or the more severe the exercise the more prominent become factors 2, 3, and 4, the greater the rise of blood-pressure and the greater the pulse-rate. The slowness at which conditions return to normal is more or less proportional to the exertion and the fatigue.

It is also true that for a given amount of exercise performed in a given time the amount of CO_2 formed is least when it is done with least effort by trained individuals and increases when the effort becomes marked.

Zuntz and Schumburg have shown upon German soldiers that a certain short march used up only 554.8 calories of energy when the subjects were fresh, but required 635.5 calories when they were fatigued. This is probably due to the fact that with the increase in effort accessory muscles are called into play, many of which contract and give off CO_2 without materially improving the execution of the exercise.

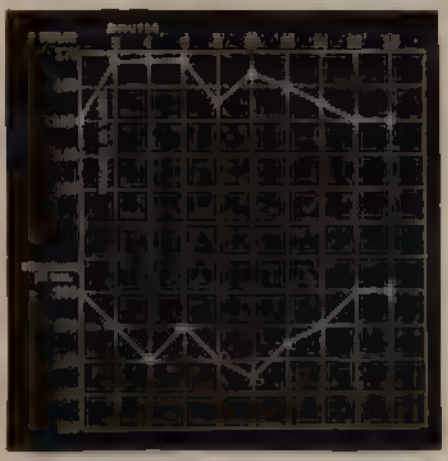

FIG. 114.—Effect of walking on a level on patient with badly broken compensation. (After Cabot and Bruce, *Am. J. M. Sc.*, cxxxv.)

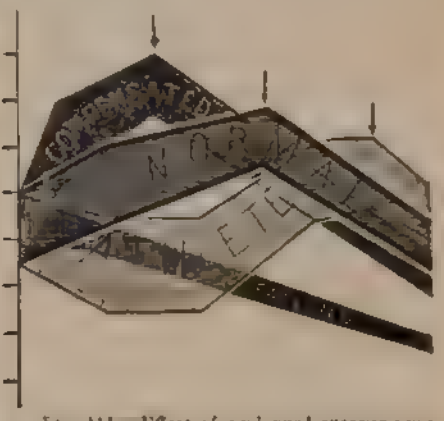

FIG. 115.—Effect of prolonged exercise upon the blood-pressure of men in various degrees of muscular strength. The arrows indicate the point at which symptoms of exhaustion set in. *COMPENSATED* compensated heart lesions, *FAILING*, broken compensation with heart failure.

Exercises of Endurance.—The point at which an exercise of speed becomes converted into an exercise of endurance is more or less relative and depends chiefly upon the condition and the training of the individual. The most typical exercises of endurance, the forced march, the long-distance runs (Marathon races), and long-distance bicycle races, have been carefully studied by Zuntz and Schumburg, Blake and Larrabee, Dietlen and Moritz, and R. T. Abercrombie. In these exercises the least changes occur in the best-trained individuals in whom the amount of effort put forth is least or least prolonged.

The pulse-rate of the men who finished in the Marathon races at Boston showed surprisingly little increase, the greatest rise during the race of 1900 being from 76 before to 144 after, but the average rate after the race was 103 (Blake and Larrabee). There was frequently a moderate grade of irregularity. Zuntz and Schumburg found similar effects. The blood-pressure after the race was usually found to be a trifle lower than before the start, though it varied greatly in different individuals. J. Baruch has recently obtained similar results with the Erlanger apparatus upon another set of trained Marathon racers. The orthodiagraph showed dilatation of the heart in all his cases. Quite different are the results in long-distance races run by amateurs. Dr. R. T. Abercrombie has recently made a careful study of the condition of contestants in a twenty-mile road race before and immediately after the race. Before the race the average blood-pressures with the Erlanger apparatus were: maximal 120–130, minimal 75–80, pulse-rate 80. Immediately after the race the pulse was in almost every instance too feeble to be counted, as were also the heart sounds, and neither these nor the blood-pressure could be satisfactorily estimated until one-half hour after the finish, when the pulse-rate was usually about 120 per minute, the maximal pressure about 75–100 mm Hg. The heart sounds were still

rapid and feeble. Nevertheless all of these men felt quite well and were able to enjoy a cold plunge immediately after the examination. Within an hour after the finish they were all feeling quite active. The blood-pressure was usually found to be lower than before the start, but this varied greatly in individual cases.

Not all the results of endurance tests are as mild as these. During the amateur athletic contests in the United States the past five years there have been several cases of permanent heart failure following directly upon overstrain in long distance runs. As in da Costa's series the persons whose hearts were injured were usually boys under twenty who were poorly trained and whose hearts were not fitted for the strain put upon them.

As regards the metabolism during such exercise Zuntz and Schumburg and also A. Loewy and L. Zuntz, found that both the amount of CO_2 given off per minute and the respiratory quotient were markedly lessened (CO_2 falling from 802.3 c.c. to 743.0 c.c. per minute, respiratory quotient falling from 0.855 to 0.780) at the end of the exertion though the O_2 used was unchanged. This is due to formation of intermediate oxidation products, sarcolactic acid, oxybutyric acid, etc., the pressure of whose salts may add to the fatigue. Moreover the lessened output of CO_2 indicates a low CO_2 content of the blood (acapnia), and as Henderson has shown, this in turn causes dilatation of the veins and causes the blood to gradually leave the arteries, stagnate in the venous reservoir (see page 31), and thus diminish the rapidity of the blood flow.

Corresponding to the variations in rapidity of blood flow, the urine is increased in amount during mild exercise, decreased during severe exercise. After boat races and after the Marathon races it often contains albumin, casts, and even traces of blood, probably as a result of stasis or high pressure in the renal veins and capillaries.

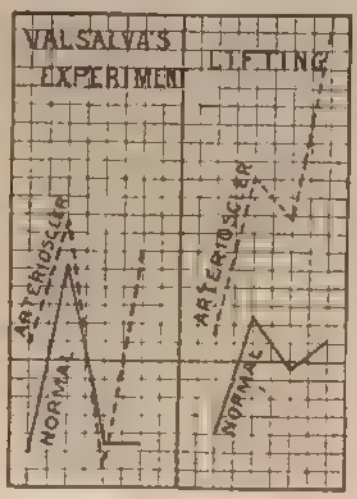

FIG. 116.—Rise of blood-pressure during Valsalva's experiment and during exercise. Normal individual. (Schematic after Bruck.) *ARTERIOSCLER*, curve of blood-pressure in man with arteriosclerosis performing the same exercise.

Exercise of Strain.—The effect of exercises of strain, lifting, etc., is totally different. McCurdy, Bruck, and others have shown that these exercises cause a far greater rise of blood-pressure than do the exercises of speed, and, on the other hand, the pulse-rate does not rise rapidly but is at first either slowed or unchanged. The rise in blood-pressure is greater in arteriosclerotics, old persons, and weak individuals for the same amount of work than in well-developed normal individuals. In persons already suffering from broken compensation, on the other hand, the heart absolutely fails to respond with increased effort, and may be so greatly weakened by the strain that the blood-pressure may fall.

All the factors which are called into play by the hormone action of CO_2 in exercises of speed and of endurance are also acting in exercises of strain; but, since the latter are usually intermittent or of short duration, their effects are at first overshadowed by others which are more intense.

In carrying out any exercise involving muscular strain the individual involuntarily closes his glottis

and executes an attempt at forced expiration. The result of this is a tremendous increase in intrathoracic pressure, which hinders the outflow of blood from the right ventricle as well as the inflow into the right auricle.

The result of these two factors is dilatation of the right ventricle and stasis in the systemic veins, which is still further shown by the cyanosis of the face and distention of the veins that accompany all such exercises even in trained athletes. The venous stasis is further increased by the sudden squeezing out of blood from the large masses of skeletal muscles, which are being forcibly contracted simultaneously, as well as from the vessels of the splanchnic area.

The high pressure within the lungs stimulates the sensory endings of the vagus, which in turn reflexly stimulate the motor nucleus of the vagus and the vasomotor centre in the medulla and cause both slowing of the pulse and rise of blood-pressure. The general result is the same, but less marked when the Valsalva experiment only (forced expiration with glottis closed, is carried out, and depends very largely upon this factor.

SIZE OF HEART AFTER EXERCISE.

Diminution in Size in Healthy Hearts. Examined with the X-ray the auricles are seen to dilate greatly, but the ventricles do not, as a rule, show any dilatation whatever. This again is a question of tonus, and here also the latter factor seems to determine whether dilatation shall set in or not. All exercises when sufficiently severe lead to dilatation of hearts whose myocardium has suffered injury, especially during the course of

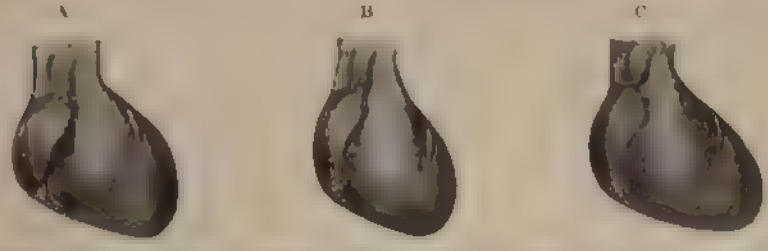

Fig. 117.—Semi-schematic drawing showing variations in size of the heart of a long-distance bicycle rider as the result of a very long race; reconstructed from the orthodiagraphic outline. A, Before the race. B, Immediately after the race, showing the great diminution in size of the heart. C, Four weeks later. After Moritz and Diethen, *München. med. Wochschr.*, 1908, ii.

infectious diseases (da Costa, Zuntz and Schumburg, de la Camp, Moritz and Diethen) or during the first few weeks following them. On the other hand, Schott has claimed to have seen cardiac dilatation in healthy wrestlers and bicycle riders as a result of short wrestling bouts. This fact has been disputed by a number of observers who have carefully controlled the more or less subjective findings of percussion by outlining the heart with the orthodiagraph.

The following exercises have been studied: bicycle riding, by Mendelsohn, Albu, Bever, Schoffer, Diethen and Moritz; marching, by Zuntz and Schumburg, Albu and Caspari, Balders, Heubelheim and Metzger; football playing, by F. Pick and by Selig; ski running, by Benischen; wrestling, by Levy-Dorn, Selig, Mendl and Selig; swimming, by Kienbock, Selig and Beck.

The results of these observations quite uniformly confirm those of de la Camp in showing that exercise, even to the point of exhaustion and

fainting, does not bring about cardiac dilatation in otherwise healthy men. In most cases the X-ray and orthodiagraph show an actual diminution in the volume of the heart [1] (see Fig. 117). De la Camp also found that healthy dogs could run upon a tread-mill until they dropped from exhaustion without causing dilatation of the heart; whereas the hearts of dogs which had been poisoned with phosphorus and which were in a state of mild fatty degeneration dilated greatly from the same exercise.

Dilatation and Myocardial Injury. — On the other hand, Hornung, who has watched the course of 1100 cases of weak heart with the X-ray, states that in such persons acute dilatations (demonstrable with the orthodiagraph) are very common as the result of slight overstrain. It may require comparatively little strain to bring this about. For example, he cites the case of a woman with a weak heart who acquired a dilatation by taking a short cut instead of a gradual ascent while climbing a hill (Oertel's Terrainkur). The dilatation lasted for several days and gradually passed off. Persons whose hearts are in this labile equilibrium are liable to have repeated attacks. But the cardiac condition rarely stands still. It gradually becomes either better or worse, according to the treatment and the mode of life of the patient.

Thoracic and Abdominal Constriction as a Factor in Cardiac Overstrain. — A high diaphragm due to tight belts or corsets is one of the most important factors which predispose to cardiac overstrain. This was already shown by A. R. B. Myers in 1867. Myers observed that certain cavalry regiments in the Indian Army were particularly subject to cardiac overstrain and to chronic cardiac disease, even more so than the infantry regiments which were doing more arduous work under the same conditions of climate and diet. He noticed that the uniforms of this cavalry regiment were very tightly belted and had tight cuirasses compressing the chest. Upon experimentation he found that the men in this regiment, when not wearing their uniforms, were quite as strong as those of other regiments in the service. He also found that the same men were able to withstand much greater exertion in the same uniforms if only the belts were worn looser. This has been shown with somewhat greater exactness by Th. Schott. Schott demonstrated with the orthodiagraph that wrestlers could withstand much greater exertion before the onset of acute dilatation or of cardiac symptoms if they wore no belts than if they were tightly belted. This is, of course, not surprising, and is simply another way of demonstrating the every-day experience of most healthy women that they can do more work without a corset or with a loose one than when wearing one that is tightly laced.

The reason for this is twofold. The belt interferes with the respiratory movements of the abdomen and diaphragm, and hence diminishes the rhythmic alternation of positive and negative pressures, of force-pump and suction-pump action, in one of the largest of the vascular reservoirs, thus

[1] The syncope (cerebral anæmia) under these conditions is probably due to the exactly opposite condition, diminished cardiac filling and hence diminished cardiac output; arterial anæmia due to rapid pulse-rate in a heart whose tonicity is increased. (This condition is fully discussed in the chapters on Paroxysmal Tachycardia and Miscellaneous Heart Diseases.)

diminishing the rapidity of blood-flow. Moreover the viscera are pushed back and the diaphragm is pushed upward by the belt, and this causes the heart to assume a more transverse position, in which kinking of the great veins, the aorta, and the pulmonary artery sets in, and both the filling and the emptying of the heart are impeded. This mechanism is readily demonstrated upon the exposed heart of the living animal. A comparatively slight upward or downward displacement of the heart from its natural position may cause tremendous fall in blood-pressure and interference with the work of the heart.

PHYSIOLOGICAL FACTORS BRINGING ABOUT DILATATION.

The diminution in the size of the heart which was found so uniformly by the above-mentioned observers seems to be due, in part, to diminished filling of the ventricles when the heart is rapid, but chiefly to the fact that the cardiac tonicity was increased by the strain.[1] This clinical observation has its analogue in experimentation on animals. O. Frank has shown that, other things being equal, a moderate increase in intraventricular pressure acts as a stimulus and causes an increase in the force of the next beat. If the pressure is raised further it reaches an optimum; but if it

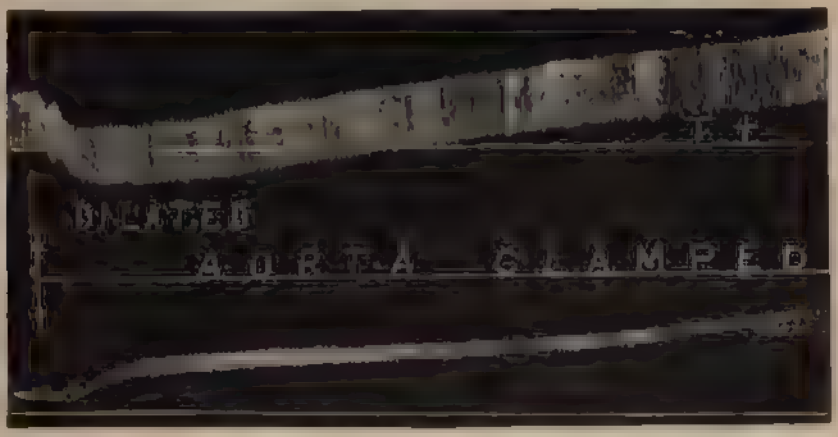

FIG. 118. — Effect of strain upon the dog's heart whose tonicity is good. Volume curve (*VOL.*) and blood-pressure curve *B. P.* of an animal whose heart is in good condition. Descending thoracic aorta compressed at the moment indicated by the arrow. Momentary dilatation followed by a diminution in size. The heart becomes smaller than before the clamping. Tonicity is increased. *T.* Blood-pressures maximal and minimal are also increased.

becomes too high the force of contraction becomes much weaker than if there were no load at all. There is a similar effect upon cardiac tonicity. Hirschfelder has shown that if the thoracic aorta of the dog is clamped the ventricles at first dilate rapidly and the systolic output diminishes. If the heart is in good condition the systoles soon begin to increase, the excess of blood is pumped out of the ventricular cavities in systole, and on the other hand, in spite of the high pressure in the veins, less blood enters the ventricles than before.

[1] It is possible that acapnia (page 31) may play a rôle under these conditions.

The amount of blood which enters the ventricles depends upon two factors: 1. As Howell and Donaldson have shown for the excised heart, and Roy and Adami for the dog's heart *in situ*, it is more or less proportional to the venous or intra-auricular pressure. 2. Roy and Adami, Hirschfelder, Cameron, and others have shown that it is also dependent upon the cardiac tonicity, being greater when tonicity is low (dilatation) and least when tonicity is high. It is therefore evident that a heart whose tonicity is high will withstand a comparatively high venous pressure without dilating, whereas when the tonicity is low it readily overfills.

Several factors contribute toward diminishing the strength of an overfilled heart: 1. With the increase of the cubical contents and the internal surface of the ventricles the mechanical work necessary to exert a normal

FIG. 119.—Volume curve of a dog whose cardiac tonicity is low. Clamping the aorta is followed by permanent dilatation and only a slight momentary increase in blood pressure. The systolic output is diminished, owing to inability of the heart to force the usual quota of blood against the increased resistance.

pressure is increased (Roy and Adami). 2. In the dilated heart the blood flow through the coronary arteries and hence the nutrition of the cardiac walls is diminished (Hyde). Moreover the dilatation of the ventricles may or may not be permanent, dependent upon the tonus of the heart muscle (Hirschfelder, Cameron). If the latter is low the dilatation remains and increases, whereas if it is high the increased pressure acts as a stimulus. It is usually a high venous pressure which keeps the heart dilated and a low tonicity which permits it to remain so.

Since the venous pressure is certainly highest in the exercises of strain, it is not surprising that permanent heart trouble arising in previously healthy persons as a result of primary cardiac overstrain is particularly common among persons (butchers, porters, stevedores, etc.) who lift the heaviest weights.

EFFECT OF THE STRAIN UPON THE HEART.

The response of the heart to a muscular exertion which just fatigues may be of three grades: 1. The heart becomes smaller or the cardiac outlines are unchanged—tonicity high (normal hearts). 2. There is a transitory dilatation (after acute infections and in hearts with myocardial or some other cardiac disturbance). 3. The overstrain leads to permanent injury of the heart, often with permanent arrhythmia (chronic car-

diac overstrain, myocardial changes). These three conditions find their analogues in the effects of clamping the thoracic aorta upon the volume of the ventricles (Fig. 119).

As has been seen in the cases quoted above, the repetition of the strain is quite as important a factor in heart failure as is the overstrain itself. Even a heart with extremely low tonicity will, in most cases, recover and gradually return to normal volume after the strain has been removed, but during the period when it is still dilated it is much more susceptible to a further overstrain. On the other hand, after a sufficient period of rest it regains its former volume and still later its former tonicity, and once more reaches its original strength. That this is probably the case in man also is shown by the fact that Poynton did not regard an occasional overstrain

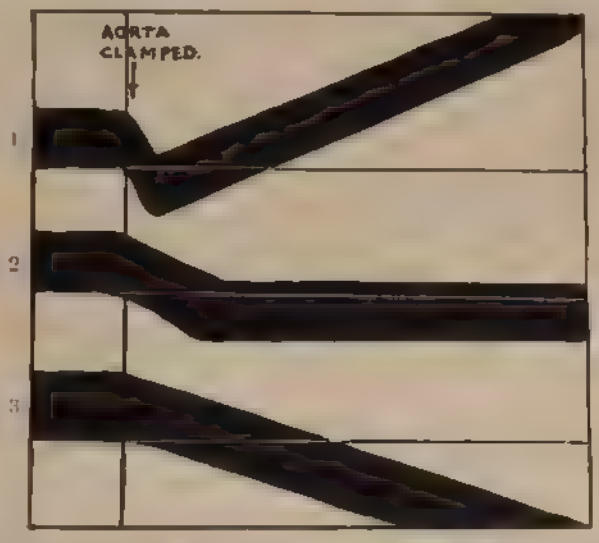

Fig. 120. Effect upon the volume of the dog's heart produced by clamping the descending thoracic aorta. Ascent of curve [illegible] in volume, descent [illegible]. 1, normal and vigorous heart; 2, slightly weakened heart with diminished tonicity; 3, very weak heart with much diminished tonicity.

as of any special significance in boys, provided it were followed by a period of sufficient rest. Moreover, Meylan has found that the lives of oarsmen upon the Harvard boat-crews were somewhat longer than the average for normal individuals, in spite of the fact that albuminuria and other signs of cardiac overstrain are quite common just after such races. Indeed not a single case of cardiac disease developed among the 152 oarsmen of his series, which comprised members of the intercollegiate crews from 1852 to 1892. There was only one case in which enlargement of the heart was noted and one case of irregularity, but neither of these inconvenienced the patient.

These individuals, in contrast to cases like that of v. Leyden, had rested sufficiently between the periods of strain, and the second strains had not been imposed upon their hearts until long after their strength and tonicity had returned to normal. In v. Leyden's case and other cases of permanent

heart failure, the heart was still dilated at the time of renewed strain. This condition seems to determine the border-line between heart failure and recovery.

The border-line conditions may therefore be summarized as follows: Dilatation of the heart during or after exercise represents a pathological, though not a very infrequent, condition in which the heart has overstepped its limits. The condition usually recedes and leaves no traces unless the heart is again overstrained while still in a dilated condition.

As regards the anatomical changes induced by the condition of overstrain, Roy and Adami have shown that, when the dog's heart begins to fail after clamping the aorta, stasis occurs in the coronary veins and the heart muscle becomes œdematous. This œdema is especially marked in the regions which are richest in connective tissue, the auricles and the auriculoventricular valves. They believe that when the strain is continued the œdema is replaced by infiltration, the infiltration by connective tissue, and that fibrous myocarditis results. Indeed, a fibrous myocarditis (cardiosclerosis) is a common autopsy finding in cases of long-continued cardiac overstrain in which there has been no severe infectious disease to account for the lesion. On the other hand, Pearce and Fleisher and Loeb have found exactly the stages mentioned by Roy and Adami in animals in the various stages of adrenalin myocarditis (see page 226).

BROKEN CARDIAC COMPENSATION

As long as the heart is able to maintain a certain velocity of blood flow throughout the circulation, the latter may be said to be compensated, but when the blood stagnates to such a degree as to give rise to the signs and symptoms of stasis, compensation may be said to be broken.

There are two forms of broken compensation. When the blood stagnates in the systemic veins from failure of the right side of the heart, the condition may be termed *broken systemic compensation*, when stasis occurs in the lungs because the left side of the heart is not acting as strongly as the right, *broken pulmonary compensation* results. Each of these two forms brings with it a characteristic group of symptoms: The broken systemic circulation (usually designated simply as "broken compensation") manifests itself in the signs and symptoms which are seen in tricuspid insufficiency—breathlessness, cyanosis, œdema, beginning in the feet and legs, enlargement of the liver, and systolic pulsation of the liver and veins, etc. Broken pulmonary compensation is accompanied by the signs and symptoms of an acute severe mitral insufficiency—intense respiratory disturbance, dyspnœa, cough, occasionally pulmonary hemorrhage, and the sputum containing the characteristic cells of passive congestion (Herzfehlerzellen).

Broken Systemic Compensation.—From the physiological stand-point, the cardinal features of broken systemic compensation are dilatation and weakening of the right ventricle, dilatation and paralysis of the right auricle, increase in CO_2 and decrease in O_2 in the venous blood, functional insufficiency of the tricuspid valve, rise in venous pressure (often to as high as 20 mm. Hg) (Fig. 121, III). The signs are cyanosis, engorgement and systolic pulsation of the veins, enlargement of the liver, œdema of the

feet and legs, and sometimes venous stasis in the medulla, vasoconstriction, high blood-pressure, and dyspnœa of medullary origin.

Broken Pulmonary Compensation.—The characteristics of broken pulmonary compensation are dilatation and weakening of the left ventricle, dilatation and usually paralysis of the left auricle, rise of pressure and stasis in the pulmonary veins, engorgement of the pulmonary capillaries, and "erection" of the lung tissue (v. Basch) (Fig. 121, IV). Welch has shown that when the stasis is very intense, pulmonary œdema sets in. V. Basch and his pupils have applied this idea to the milder pulmonary manifestations and have shown that a moderate erection of the lung tissue brings on cardiac dyspnœa and leads to bronchitis and cough. His pupil, Kauders, has shown that the position of the diaphragm is affected reflexly by the amount of blood in the lungs, congestion causing the diaphragm to descend, depletion causing it to ascend. It is thus usually lower than normal in mitral lesions, higher in pulmonary and tricuspid.

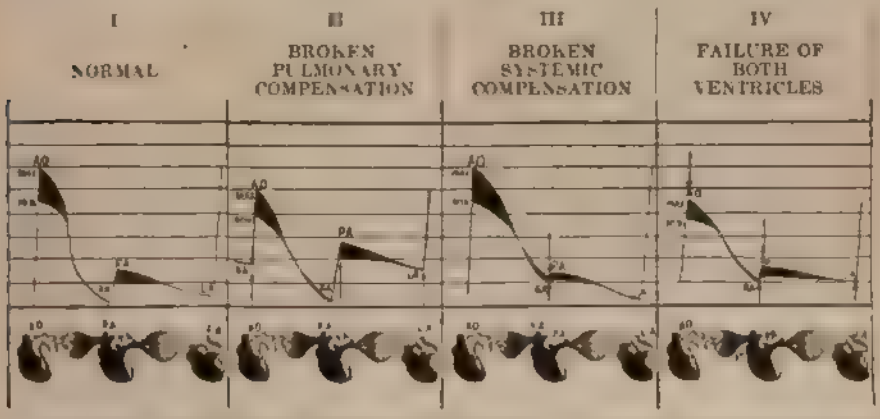

FIG. 121.—Diagram showing changes in the circulation. I, normal. II, broken pulmonary compensation. III, broken systemic compensation. IV, both compensations fail, stases in lungs and veins. *AO*, pressure in the aorta. *PA*

V. Basch also believed that the congestion of the lungs causes the elasticity of the lungs to diminish and to become so rigid as actually to diminish the respiratory expansion, but the experiments of D. Gerhardt have thrown doubt upon this phase of his conclusions. As regards the changes of pressure and the distribution of the blood, however, v. Basch's conclusions have been confirmed, not only by Gerhardt in Germany but by W. G. MacCallum and McClure in America.

In badly weakened hearts both forms of broken compensation may be present, sometimes features of one, sometimes of the other, predominating.

Functional Valvular Insufficiency in Broken Compensation.—Although it has not been absolutely proved, it seems almost certain that the occurrence of broken compensation from acute dilatation is accompanied by a functional insufficiency of the tricuspid or the mitral orifice which may be of transitory duration. Indeed this functional insufficiency of the tricuspid valve in heart failure is much more common than organic lesion of

the valve, and in long-standing cases is accompanied by actual stretching of the tricuspid orifice (T. W. King, G. A. Gibson, Mackenzie, Keith).

T. W. King, in 1837, demonstrated that such functional insufficiencies occur at the tricuspid valves, and even that they were dependent upon the tonicity of the ventricular fibres; since the valves which had been insufficient a few hours after death held water perfectly after rigor mortis had set in (quoted in full on page 396). These observations have been confirmed and extended by G. A. Gibson, François-Franck, Mackenzie, Friedreich, Marey, Hirschfelder, Keith. Hering demonstrated the same phenomenon for the mitral valve in rabbits, but found that in dogs the mitral valve did not leak even after clamping the aorta. Stewart and the writer have been able to demonstrate the occurrence of such an insufficiency of the mitral valve when the aorta was clamped, in dogs whose aortic valves had been rendered insufficient. In man Morton Prince and Broadbent have noted the presence of transitory mitral systolic murmurs (sometimes transmitted to the axilla) in men who were being subjected to the strain of civil service examinations, and in cases with similar signs Minkowski has obtained tracings from the œsophagus which have the form characteristic of mitral insufficiency.

In the earlier stages of cardiac overstrain the dilatation of the auricles is a more or less passive phenomenon which exerts little influence upon the circulation, but in the more severe stages it may play a leading rôle.

Auricular Paralysis and Arrhythmia in Cardiac Overstrain. — Conditions which affect tonicity and filling of the ventricles have a still greater effect upon the tonicity and filling of the auricles. It was demonstrated by Ludwig's pupils, Waller (1878) and v. Frey and Krehl (1890), that when the ventricles began to fail, the auricles soon became overloaded with blood and ceased to contract entirely when the pressure reached 15–20 mm. Hg. As a rule this does not affect the cardiac rhythm, but Hirschfelder has shown in dogs that when this is brought about by narrowing the mitral orifice an absolute irregularity (disorderly rhythm) may set in without any apparent contractions carried out by the auricles.

It seems probable that, under these circumstances, the contraction stimuli originate in the auricle and are transmitted but are not carried out by these chambers, just as Biedermann has shown that muscle in water rigor can originate and transmit a stimulus without itself contracting. Mackenzie and others believe that under these circumstances the stimulus no longer originates in the sinus portion of the auricle but in the Purkinje cells of the His bundle (at Tawara's nodal point, Knotenpunkt) and hence designates this absolute irregularity as nodal rhythm. They believe that the auricle and ventricle are contracting simultaneously under these circumstances. Since Retzer has traced the bundle directly into the sinus region and believes that there is no nodal point, this view seems open to question.

Arrhythmia. — Whatever may be the origin of the arrhythmia it is very common in severe overstrains. This not infrequently arises in the course of valvular lesions as well as Mackenzie has proved. The case cited on page 550 gives an example of such an irregularity arising during such an attack and subsiding a few days later after rest and digitalis. Five days later the rhythm became regular and the auricles were contracting once more. When the overstrain is more protracted the auricular contraction may remain absent for weeks and even months, and most frequently if it has persisted for a considerable length of time, permanent changes set in in the musculature of the sinus region (Keith, Schönberg) and regularity is never regained. The pulse has become permanently irregular (pulsus irregularis perpetuus, arrhythmia perpetua). As has been seen on page 77, the arrhythmia

itself slows the blood stream and the diseased condition of the sinus prevents the heart from compensating for this by a greater number of contractions. The velocity of the circulation is thus self-limited. Only a certain amount of CO_2 per minute can be taken care of and any excess brings on overstrain.

Changes in Venous Pressure.— Changes in pressure in the systemic veins, which show how well the right ventricle is pumping often afford an excellent index of the break in systemic compensation, rising from normal pressure of 5–10 cm H_2O to a height of 20 or 25 cm. It usually rises when the patient's condition becomes worse and falls as improvement sets in (Hooker and Lyster).

The arterial pressure, on the other hand, is affected by too many factors to show characteristic changes. It may be kept up until shortly before death, by asphyxia of the medullary centres and resultant vaso-constrictor and augmentor stimulation; or, on the other hand, when this mechanism is not brought into play, the arterial pressure may be low and the pulse may be small and weak.

CARDIAC FAILURE WITH A SMALL HEART.

There is another form of failure of the circulation which sometimes occurs as the result of exertion, even in trained athletes. This form is accompanied by pallor, a small rapid pulse, and sometimes even by syncope. However, as Dietlen and Moritz have shown, it is not accompanied by a dilatation of the heart but, on the contrary, the latter is smaller than normally. It is a failure of the rest of the circulation rather than of the heart.

It must be admitted that this condition has not attracted much attention, and but little can be said of the mechanisms involved. The pallor, small pulse, and small heart, however, are features which are also common to the condition of shock and the cardiac neurosis. In these conditions, the important mechanical factors are the accumulation of blood in the dilated abdominal veins, giving rise to a low venous pressure, the diminished filling of the heart, and consequently the diminished output into the aorta. The symptoms are symptoms of "arterial anæmia."

The causal factor in bringing about this condition may be dilatation of the veins. In the case of exercise this vaso-dilatation may result either reflexly from disturbed digestion, or, perhaps, as Henderson suggests for somewhat similar conditions, it may set in when the rapidity of breathing exceeds that necessary to aerate the blood, even to meet the increased needs of the body. Under these conditions CO_2 leaves the lungs, and hence also the blood, a little too rapidly, acapnia results, and, as its first effect, allows the veins to dilate (see page 31). The blood thus stagnates in the veins. As a matter of fact, Krogh, Zuntz and Schaumburg, and also A. Loewy have shown that at this stage of exercise less CO_2 is given off from the lungs than before, and the respiratory quotient CO_2 is lessened. They believe that oxidation is less at this stage and hence less CO_2 is present in the blood. In other words, from a totally different stand-point, and years before Henderson's experiment, it was rendered probable that a state of acapnia is present at the stage of fatigue in excesses of endurance, and therefore that the mechanism which he observed to be active in acapnia is largely responsible for this form of circulatory failure.

FUNCTIONAL TESTS OF CARDIAC EFFICIENCY

It is evident from the facts discussed above that the most important question in the functional study of heart failure is to determine accurately the border-line between fatigue and overstrain, to distinguish between the normal and the pathological. Various tests have been devised for this purpose.

1 **Postural Change in Pulse-rate.**—The rise in the pulse-rate which occurs when the patient stands after lying down is of some importance. Under normal conditions the acceleration is not more than twenty beats per minute, the average acceleration for normal individuals being seven. However, this depends upon many factors, one of which is the length of time during which the patient has lain down, his state of mental excitement or quiet, etc. The psychic element plays a particularly important rôle in this test.

2 **Contraction of Antagonistic Muscles.**—Herz has introduced another procedure, the self-checking or self-antagonizing test (Selbsthemmungsprobe). He counts the pulse over a period long enough to assure a reasonably constant rate per minute. The patient is then made to sit down and very slowly flex and extend the right forearm, putting all the while his full attention upon the movement, but contracting simultaneously the flexor and extensor muscles of the arm, and attempting to antagonize his own movement with as much force as possible. This converts the exercise into a mild exercise of strain. Herz states that in normal individuals this causes no change in pulse-rate, while in those with feeble hearts the pulse-rate is slowed 5–20 beats per minute. (Perhaps this is due to the more vigorous expiratory effort which accompanies this procedure in persons with diseased hearts.) Cabot and Bruce have repeated Herz's observations, and find that they are correct in at least a certain number of cases, but they are unwilling to subscribe to his general rule. The writer also has found a number of perfectly strong and healthy individuals who give Herz's pathological reaction.

3 **Rise of Blood-pressure on Constricting the Femoral Arteries.**—Marey (1881) demonstrated that in normal individuals the blood-pressure rose when both femoral and both brachial arteries were compressed. Katzenstein found that on compressing both femoral arteries alone, in

	Blood-pressure	Pulse-rate
Normal individuals	Rose 5–15 mm.	Fell.
Compensated cardiac lesions	Rose 15–40 mm.	Unchanged or fell.
Slight cardiac insufficiency	Unchanged	Unchanged or rose.
Very weak hearts	Fell	Fell.

Hoke and Mende and others have repeated Katzenstein's observations, and find that, though these results hold true in general, the method is unreliable as a test and in bad cases is too dangerous for use.

4 **Rise of Blood-pressure upon Exercise.**—Another method, introduced by Gruupner, of Nauheim, depends upon the rise of blood-pressure which occurs during exercise. Gruupner found that, as Masing had shown, mild rapid exercise, such as walking up and down stairs rapidly, etc., caused a rise of blood-pressure in normal individuals but a fall of pressure in those with failing hearts. His observations have been repeated on a considerable series of patients by Baur (also of Nauheim). Baur used the stationary bicycle as a test, regulating the effort by applying a loaded brake to the wheels. He found that in normal individuals there was at first a rise of 5–10 mm. Hg and later a fall of 5–10 mm., while in insufficient hearts there was a fall of 5–20 mm. Hg. The limit of performance of the latter was 45–300 Hg of work, however only a small fraction of that which could be done by the normal individuals. Cabot and Bruce also have repeated and confirmed Gruupner's observation, and believe that it will prove of assistance as an aid in functional diagnosis.

That a close relationship exists between the increase in blood-pressure and the increase in tonicity (stimulation of augmentor fibres), which results from strain put upon the heart, may be seen from the curves of Hirschfelder and Cameron in the dog's heart (quoted on page 135, and shown in Figs. 118 and 119). It is probable that, in most cases, rise of pressure corresponds to increased systolic output and concomitant increase in tonicity. It must be realized, however, that in some cases the rise may be secondary to stimulation of the vasoconstrictor centre from medullary stasis or asphyxia, but may represent an unfavorable condition.

Several objections may be made to the value of this test.

1 G A. Gordon in G A. Gibson's clinic and also Professor Dawson, in collaboration with Professor Eyster and also with Mr Hetfield, have shown that the blood-pressure in trained athletes falls during mild exercise exactly as it does in broken compensation, also that it falls when the "second wind" is acquired and while the person's functional power is increasing rather than decreasing

2 As already shown by Masing, the greatest rises of blood-pressure occur in old and feeble persons, whom the exercise brings near to the border-line of cardiac overstrain

3 In persons in whom the fall in blood-pressure occurs as a result of the test exercise, the general symptoms, respiratory distress cyanosis, etc , to say nothing of the diminished decrease in the size of the pulse, tachycardia and arrhythmia resulting, are more than sufficient evidence that the patient's strength has been overtaxed

4 These simpler clinical manifestations are more delicate indices and are less ambiguous signs than are the changes in blood-pressure

The recent studies of Schott, de la Camp, v Criegern, Hornung Moritz and his pupils, taken in conjunction with the physiological experiments of Frank, Hirschfelder, and Cameron, indicate that the only true numerical criterion of cardiac efficiency is whether a given strain causes it to diminish in size (increase in tonicity = stimulation) or to dilate (decrease in tonicity = overstrain)

Functional studies upon the border-land between functional sufficiency and cardiac failure are of the most fundamental importance, and all the facts added to our knowledge of the subject are of the greatest value in adding to our understanding of the subject.

Observation versus Estimation.—However, it must be admitted that, in order to be decisive, all these tests usually have to be pushed to a point at which the appearance, sensations, and signs of the patient are in themselves perfectly characteristic of cardiac insufficiency, and at which, for diagnostic purposes, a little common-sense observation is at least as unambiguous as observation with elaborate apparatus. This does not mean that exercise tests are unimportant. On the contrary, they are of the greatest value; and no change in the patient's mode of living during convalescence or during after life should be undertaken without them. But their importance depends more upon the care with which the physician watches the general appearance and condition of the patient, the rapidity with which he recovers from the exercise, his general condition, and whether nervousness, irritability, cough, or insomnia have set in during the twenty-four hours following it, than in the numerical changes which occur at the moment of exercise. The symptoms to be looked for as evidence of overwork are discussed in more detail in the instructions for giving Schott exercises (page 195). These are subtler manifestations resulting from smaller changes than may be detected by even the most refined observations by mechanical methods, and which are less easily masked by ambiguities. Moreover, it must be realized that any one form of exercise furnishes data which may depend as much upon the condition of the skeletal muscles as upon the heart. The blacksmith with a diseased heart may be able to do more work than the book-keeper with neurasthenia, and yet under the conditions in which he lives, even if not under the strength test arranged for the average man, the blacksmith's heart may be failing.

Relation of Functional Test to Mode of Life.—In diagnosis, prognosis, and therapy, the testing of functional insufficiency is a matter of sociology

as well as physiology. The important question is not what the patient can do in a gymnasium, but what he can do and what he can not do in every-day life. Each man must be fit for his own mode of life or must be made to change it. His cardiac power must be studied with reference to that mode of life rather than with reference to a rigid scheme.

Probably the most thorough system of routine functional testing ever instituted was that resorted to by J. M. da Costa during the Civil War before he permitted his convalescents from cardiac overstrain to return to active duty with their regiments. He subjected them first to light camp duties, then to guard duty, then to provost duty, and later made them run frequent races comparable to charges upon a battlefield—each test commensurate with the mode of life which the patient was about to live. Step by step he ascertained the endurance of his patients without overstraining them, and thus obtained a series of permanent cures which stands as a worthy monument to one of the most careful and brilliant of American clinicians.

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III.

SYMPTOMS OF CARDIAC DISEASE.

The symptoms for which a patient suffering from cardiac disturbance presents himself to the physician are variable, and frequently seem so far removed from the site of disease as to mask their real origin and even to lead to mistakes in diagnosis. Accordingly, it is important to consider the symptoms early and the conditions giving rise to them, remembering that in general they are due to (1) slowing of the circulation and local anæmia or accumulation of CO_2; (2) overfilling of the veins with blood; (3) disturbances in the nervous system, of circulatory origin.

The following are the main symptoms associated with cardiac diseases: (1) shortness of breath; (2) cough; (3) swelling of the feet and legs; (4) urinary disturbances; (5) palpitation of the heart, precordial pain, pain down the arms; (6) digestive disturbances, indigestion, vomiting, abdominal pain, jaundice; (7) general nervousness, occasionally transitory delusions and hallucinations, sometimes fainting spells.

All these symptoms are liable to be increased after exercise, excitement, or worry, on account of the increased heart-rate and often increased blood-pressure that accompany them, and the consequent increase in the work of the heart. They are also much increased by damp and sultry weather when there is little air stirring. The patient's color is then usually pale, showing a vasoconstriction to which some of the embarrassment is probably ascribable. The symptoms usually improve when the weather becomes clear.

RESPIRATORY DISTURBANCES.

DYSPNŒA.

Shortness of breath is usually the earliest and most common sign of cardiac failure and especially of failure of the left ventricle (broken compensation). The foundation for this doctrine was laid by the classical experiments of Welch under Cohnheim's direction. Welch demonstrated that in conditions in which the force of the left ventricle was impaired without impairment of the right, œdema and congestion of the lungs set in.

V. Basch and his pupils, Grossman, Bettelheim, and Kauders, have shown — in a very careful series of investigations upon animals — that dyspnoea and the other respiratory disturbances of heart failure are due chiefly to stasis in the pulmonary capillaries and veins, and are associated with rise of pressure in the left auricle. Under these circumstances the elasticity of the lungs is probably diminished and the volume of lung tissue increases. The respiratory excursion decreases. These conditions disappear when the output of the left ventricle (and hence the suction-pump action of that chamber) in-

creases sufficiently to pump out the excess of blood from the lungs and remove the stasis. Accordingly, as is the common clinical experience, severe dyspnœa is an early sign of mitral lesions, which readily comes and goes with slight overstrains; while in aortic disease it is a sign of a severe break in compensation, and often indeed of a secondary mitral insufficiency.

This group of symptoms cough dyspnœa, cardiac asthma, pulmonary œdema pulmonary hemorrhage—constitute a group of symptoms characteristic of stasis in the pulmonary veins (broken pulmonary compensation), just as cyanosis, enlargement of the liver, and ascending œdema are characteristic of failure of the right heart. In Wilkinson King's "safety-valve action of the right ventricle" failure of the latter substitutes a state of broken systemic compensation for one of broken pulmonary compensation.

Rubow has shown by spirometric investigations that in cardiac dyspnœa there is a tendency for the lungs to assume the greatest possible volume (diaphragm descends lower and lower) just as is the case in emphysema.

According to Kraus the amount of O_2 taken up by the blood and of CO_2 given off per minute is practically unchanged in cardiac failure. The conditions are therefore practically analogous to Zuntz and Schaumburg's experiment, in which increasing the CO_2 in the inspired air caused polypnœa and caused the total amount of air taken into the lungs per minute to rise tremendously without altering the amount of oxygen taken up by the blood. It is probable that stasis in the pulmonary capillaries stimulates the vagus endings in the same way as does CO_2.

Orthopnœa. One of the most striking features of cardiac dyspnœa is the fact that it is increased in the reclining posture and relieved by sitting up (orthopnœa), although this position favors the accumulation of œdema in the legs, and, as Erlanger and Hooker have shown, impedes the total velocity of the circulation. For this fact there may be several explanations, or, more accurately, there may be several factors involved:

1. As Rubow has shown, when the patient is propped up, his liver and diaphragm descend, and there is thus more air space available in the thoracic cavity.

2. Since the head up and feet down position tends to impede the return of venous blood from the lower extremities, trunk, and abdomen it thus tends to equalize the activities of the right and left ventricles. By slowing the inflow of blood into the lungs it enables the weakened left ventricle to deplete the pulmonary capillaries more effectually than would occur if more blood were thrown into them by the relatively stronger right ventricle.

3. In the head up position the veins of the medulla drain more readily, thus diminishing venous stasis in the medulla and consequently also diminishing the effect of direct CO_2 stimulation of the vagus and respiratory centres. This effect of the erect position in diminishing the volume of the blood in the brain is well known to brain surgeons, who sometimes make use of it for penetrating to otherwise inaccessible places.

Cardiac dyspnœa is particularly marked during sleep, partly because the diminished sensitiveness of the respiratory centres allows CO_2 to accumulate (if only momentarily) with greater ease than during periods of wakefulness, and partly because the muscles of the larynx relax, the laryngeal slit is narrowed, and air enters the lungs with more difficulty, thus allowing a slight asphyxia to set in.

Cardiac Asthma.—Occasionally the respiratory distress takes the form of a definite paroxysmal dyspnœa or cardiac asthma. These paroxysms

are particularly common in aortic insufficiency or coronary sclerosis. They frequently occur at the moment of awakening; or, more accurately, the patient is awakened by the need of air. CO_2 having accumulated during sleep, owing partly to the slower respiration partly to the above-mentioned relaxation of the laryngeal muscles. This factor may also give rise to an acute failure of the left ventricle from impaired cardiac tonus which arises during the mild asphyxia that has preceded. Perhaps the dyspnœa is due partly to heart failure and partly to respiratory failure.

While the patient is awake such attacks are sometimes brought on by the act of defecation. This is not surprising, since defecation presents a typical Valsalva's experiment, in which, as previously shown, a great strain is thrown upon the left ventricle, sometimes severe enough to produce a functional mitral insufficiency.

Morphine and Strychnine in Cardiac Dyspnœa.—Such attacks of cardiac asthma may be relieved by morphine, but the continued use of morphine for this purpose often has a bad effect. Though it momentarily relieves the distress, it also diminishes the irritability of the respiratory centre and thus allows still more CO_2 to collect in the blood. The patient then requires still more morphine to quiet him, and a vicious circle is introduced:

Accumulation of CO_2 in the lungs → Paroxysm of dyspnœa

↑ ↓

Diminished irritability of respiratory centre ← Morphine

On the other hand, the patient so quickly acquires the morphine habit that frequently he brings on a paroxysm of dyspnœa voluntarily in order to get the drug, and does himself considerable harm by this effort.

Under these conditions strychnine is the drug indicated by its pharmacological action in stimulating the respiratory centre (as Eyster has shown). Where strychnine (.002 to .005 Gm. = gr. $\frac{1}{30}$ to gr. $\frac{1}{12}$) or with atropine (.0005 Gm. to .001 Gm. = gr. $\frac{1}{120}$ to gr. $\frac{1}{60}$) does not suffice, morphine may have to be given, but it is best to give some strychnine along with it. After the first dose it is frequently possible to obtain the quieting psychic effect by injections of distilled water or of strychnine alone without giving rise to the morphine habit.

Cardiac Asthma from Nasal Disease. Another form of asthma with cardiac symptoms has its origin not within the heart but in the nose. Francis-Franck in 1889 was able to demonstrate that cough, laryngeal spasm (false croup), asthma, and a reflex bronchitis arise reflexly from stimulation of the nasal mucosa. He was able to reproduce these phenomena in animals by stimulating the mucosa of the septum. They were accompanied by acceleration of the heart and vasoconstriction. They did not appear when the latter had been cocainized, or if they had once set in, they disappeared on cocainization. He found that these effects were much more pronounced in animals with experimental aortic insufficiency than in normal animals, and he believes that such exaggerated responses to nasal reflexes are responsible for many of the paroxysmal respiratory disturbances in cardiac patients suffering from cardiac disease, and especially from aortic insufficiency. It is possible that they may give rise to some of the vasomotor crises.

It is important to differentiate cardiac asthma from the bronchial form. Both may be accompanied by bronchitis and by the presence of rales. In the cardiac form there is no impediment to either expiration or inspira-

tion, and hence only a simple polypnœa is observed, while in bronchial asthma there is stenosis of the smaller bronchi with hinderance to both expiration and inspiration, and hence a peculiar labored and wheezing breathing with prolongation of expiration which is quite characteristic.

C M Cooper (The Respiratory Ratio: A Preliminary Note, J Am M Asso , Chicago, 1909, lii, 1182), suggests that the differential diagnosis in doubtful cases may sometimes be made by noting the ratio between the periods during which the breath can be held in full inspiration and those in which it may be held in full expiration. In normal individuals the breath may be held in inspiration from 40 to 70 seconds; in expiration from 20 to 35 seconds ($i.e.$, ratio $\frac{40\text{–}70}{20\text{–}35}$) In patients with cardiac insufficiency this ratio is preserved, though the periods are shortened ($\frac{25}{15}$) In bronchial asthma, on the other hand, the breath can be held longer in expiration than in inspiration and the ratio is reversed ($\frac{15\ 25}{25\ 35}$)

It seems probable that this ratio will prove of great value in differentiating between asthmatic attacks due to acute failure of the left ventricle and those which arise in cases of cardiac diseases, from reflexes of nasal or visceral origin.

PULMONARY ŒDEMA.

Occasionally the attack of cardiac failure is very severe and is accompanied by œdema of the lungs. The above-mentioned experiments of Welch and Cohnheim, and later of v. Basch and his pupils, have shown that this is due to pulmonary stasis from acute failure of the left ventricle It is most frequent in cases of mitral stenosis in which pulmonary stasis readily sets in, and is the complication particularly to be feared during pregnancy and labor.

Experimentally, pulmonary œdema is readily produced by overstraining the left ventricle through an overdose of adrenalin (J. Loeb[1]). Besides conditions of cardiac disease it is occasionally encountered after operations in which adrenalin has been used, especially when in large amounts or upon surfaces from which it is readily absorbed Occasionally, as in a case recently known to the writer, sudden death results from this cause from an operation otherwise trivial.

J. J. Miller and S A Matthews have recently investigated the action of numerous poisons in producing pulmonary œdema, and have found that mechanical factors in the circulation (failure of the left ventricle) are the immediate cause of the pulmonary œdema after adrenalin, iodides, and iodine, but that acetic ether, nitric oxide, and ammonia cause it to appear without any evidence of disproportion between the action of the two ventricles These substances apparently act entirely by injuring the walls of the pulmonary vessels and by increasing the secretion of the alveolar walls cells, and pulmonary lymph.

Signs of Pulmonary Œdema. — The onset of pulmonary œdema is marked by pallor, cyanosis, cough, coarse moist râles throughout the chest, and often by a frothy serous expectoration which may be very profuse. Friedrich Muller has shown[2] that this expectoration contains considerable

[1] Haven Emerson has shown that this occurs only when the chest is closed, not when the chest is opened and artificial respiration substituted, and that it can often be cured by artificial respiration with bellows inflation. Barringer reports good results from artificial respiration in one patient

[2] To demonstrate the presence of albumen add dilute acetic acid to the sputum to precipitate all the mucin, filter and then precipitate the albumen from the filtrate by the addition of potassium ferrocyanide

albumen, a fact of great diagnostic importance in doubtful cases. These symptoms constitute a signal for immediate action.

Treatment.—The strain upon the left ventricle may be relieved by inhalations of amyl nitrite, and its strength may be increased within a few minutes by intravenous injection of strophanthus. The most certain procedure, however, is venesection, since it diminishes the work of the right heart at once and thus enables the left ventricle to equalize conditions. Accordingly pulmonary œdema is the signal for venesection in any except the most anæmic persons.

For the œdema itself atropine (0.5–1.0 mg., $\frac{1}{120}$ to $\frac{1}{60}$ gr. hypodermatically) should be given, inasmuch as it diminishes the bronchial secretions and thus does away with the œdema. In desperate cases larger doses should be used.

HEMORRHAGE FROM THE LUNGS.

As a result of engorgement of the pulmonary capillaries or of permanent injury to their walls, hemorrhages from the lungs may occur in any acute cardiac overstrain, but they are most frequently seen in cases of mitral disease. In itself such a pulmonary hemorrhage is of no importance, although by relieving the congestion it may bring a great deal of subjective relief to the patient. On the other hand, the conditions which bring them on acutely are frequently those of severe overstrain. They are particularly common in mitral disease, in pulmonary insufficiency and sclerosis, and congenital heart diseases

It is always of the greatest importance to differentiate between such an hæmoptysis and that of an early pulmonary tuberculosis and only the most careful repeated examinations, coupled with the presence of the cardiac lesion and the absence of signs of pulmonary disease, give sufficient grounds to exclude the latter It is particularly important to examine for tubercle bacilli in the blood spit up, since after a hemorrhage they may not be present again in the sputum for several months

Rest and the general measures which diminish pulmonary engorgement constitute the treatment.

PULMONARY EMBOLISM AND HEMORRHAGES.

Pulmonary embolism with infarction is a not uncommon complication when a clot forming in the right auricle or ventricle is loosened into the circulation and lodges in some branch of the pulmonary artery These infarcts may be large or small, dependent upon the artery occluded, and upon their size depends the severity of the symptoms and the onset of dyspnœa.

In all these cases there is an area of dulness, tubular breathing, and increased vocal fremitus over some parts of the lung, and the expectoration of a shiny, bloody or prune-juice sputum, which differs from that occurring in brown induration in being much richer, deeper in color, and in containing a great deal of more or less changed blood intimately mixed with the mucus instead of somewhat separate from it Pulmonary embolism is sometimes fatal when a large vessel is occluded, but usually not so in the first instance Exactly when a single embolus will be followed by a shower of emboli and a fatal result cannot be foretold, and the patient lives continually under a sword of Damocles, although in some cases he may live for several years without further disturbance.

CHEYNE-STOKES BREATHING.

Cheyne, of Dublin, described a peculiar form of respiration in which there are occasional pauses of variable duration, from several seconds to half a minute, followed by inspirations at first slow, then more rapid, then again lapsing into a pause. It was again described by Stokes in 1846 and is very common in bad cases. This phenomenon is particularly frequent in heart cases, especially in aortic disease, and is usually seen when the patient is asleep. It is also common in cases of brain tumor, apoplexy, or any condition where there is an increased intracranial tension. Not infrequently, in cardiac disease, the patient is in a state of coma or semicoma during the periods of apnœa but fully conscious during the periods of dyspnœa. During the former the pupils are contracted and do not react; during the latter they widen and react once more.

FIG. 122.—The two types of Cheyne-Stokes respiration in their relations to the blood-pressure curves. (After Eyster.) A. Intracranial pressure type—apnœa accompanies slowing of the pulse and fall of blood-pressure. B. The cardiac type—apnœa associated with rise of blood-pressure and quickening of the pulse-rate.

Eyster has found that the occurrence of Cheyne-Stokes respiration is always associated with the slow periodic changes of blood-pressure known as Traube-Hering waves. He distinguishes two types. In the first, which is always associated with increased intracranial tension, as in brain tumor, meningitis, uræmia, the *period of respiratory activity* is associated with a *rise* of blood-pressure and *quickening* of the pulse, the period of *apnœa* with a *fall* of *blood-pressure* and *slowing* of the pulse (Fig. 122, A).

In the second type, the common form in cardiac and arterial disease, the *respiratory activity* is associated with a *fall* in blood-pressure and *slowing* of the pulse, and the *apnœa* is associated with *rise* in blood-pressure and *quickening* of pulse-rate (Fig. 122, B). Eyster was able to reproduce the first group in animals by raising the intracranial tension and found that whenever the intracranial pressure was above the mean blood-pressure apnœa occurred. Then the blood-pressure rose through asphyxial stimulation of the vasomotor centre, and when it overtopped the intracranial pressure respirations again set in. The converse was not true of the second group of cases, and neither he nor any other observer has been able to reproduce this more common type or analyze its causal factors.

Biot has described another type of respiration, in which a series of inspirations equal in rate and in size are punctuated by long apnœic pauses. This is only a small variety of the cases showing the Cheyne-Stokes type, and its occurrence and causal factors seem to be about the same as the latter.

Mosso has also depicted another group of periodic respirations occurring at high altitudes, apparently from low CO_2 content of the blood reaching the vasomotor centre (acapnia), in which there is with each *respiratory group* *rise* of blood-pressure and *slowing* of the pulse. This does not seem to occur in cardiac or intracranial cases.

Eyster concludes that in the intracranial pressure type the Cheyne-Stokes respiration is due to the fact that the respiratory centre is more sensitive to anæmia than is the vasomotor centre, and rapidly loses its irritability, regaining it when circulation is reëstablished.

Therapeutically Eyster has attempted to remedy the condition by increasing the irritability of the respiratory centre, (1) by injections of strychnine (1.5 mg., $\frac{1}{40}$ gr.), and (2) by inhalations of CO_2. Both of these measures seem fairly successful, but more observations are necessary before conclusions can be reached. Pembrey was able to cause the periodic breathing to return to normal by causing the patient to breathe either pure O_2 or O_2 containing an excess of CO_2, demonstrating that in this case the action of the respiratory centre could be restored by either improving its condition and increasing its irritability through increased aeration, or by increasing the strength of the respiratory stimulus by increasing the concentration of CO_2 in the lung alveoli.

The occurrence of Cheyne-Stokes respiration is a very grave symptom. It is often a harbinger of death, as claimed by some authors, but the writer has seen many patients recover from it and even live for several years. It should therefore be classed with several other symptoms as indicating a grave weakening of the circulation but not necessarily an incurable one.

COUGH.

Patients with chronic cardiac trouble are very apt to suffer from a mild cough, even during their periods of remission, and especially every winter. Sometimes this may even be mistaken for a primary bronchitis. Primarily the condition is due to engorgement of the pulmonary vascular system, with increased secretion of the bronchial mucous glands as a result. It represents a state of mild loss of pulmonary compensation.

In somewhat worse form, and particularly in bad cases of chronic mitral disease, the alveolar capillary walls become so much injured that there is diapedesis of red blood-cells into the alveoli. These die and are taken up by phagocytes which find their way into the sputum in the form of "Herzfehlerzellen"—large endothelial cells containing vacuoles and numerous brown granules of hæmosiderin. The expectoration of "Herzfehlerzellen" is usually associated with a condition of brown induration of the lungs, a chronic interstitial pneumonia with dilatation of the pulmonary capillaries, tortuous condition of the vessels, and deposition of hæmosiderin in the tissues.

ACCUMULATIONS OF FLUID IN CARDIAC DISEASE.

Œdema.—When a case of heart disease reaches the stage of broken (systemic) compensation and the right heart fails to perform its function properly, œdema of the feet and legs sets in. This occurs at some stage of almost every case of heart disease, but not always at stages of equal severity, sometimes setting in quite early, sometimes only as a terminal event. Consequently, although a grave symptom, the presence of œdema need not indicate a desperate condition.

The distribution of œdema of cardiac origin differs characteristically from the nephritic type. This is readily comprehensible when it is recalled that, as Cohnheim has shown, œdema occurs only when there has been some injury to the walls of the vessels and capillaries. In nephritis Heinecke and Meyerstein have proved the existence of a substance in the blood which

injures the capillaries, and Kast has demonstrated that the blood of nephritics contains a substance with lymphagogue action. As might be expected from a poison circulating in the blood, the injury occurs simultaneously throughout the body. Hence in nephritis the œdema begins quite irrespectively of the action of gravity and is especially marked in the face and eyelids. In cardiac œdema there is no such lymphagogue poison at work, and the injury to the capillary walls is the result of local stasis, lack of oxygen in the cells of the capillary walls under the influence of the slowed circulation. Accordingly it begins where circulation is slowest, *i.e.*, in the dependent portions,—the feet and legs,—and either remains localized there, or, if the condition becomes worse, progresses upward to the genitalia, to the abdominal cavity (portal stasis), giving rise to intense ascites, to the subcutaneous tissue of the body wall, and finally to the production of fluid in the chest (hydrothorax).

Fig. 123.—Legs of a patient with extreme œdema simulating elephantiasis and tremendous ulcers. Diameter of the left leg 10½ inches (25.5 cm.). Drawn from photographs lent by Dr. W. Preston Maher, of Hagerstown, Md.)

Occasionally when the œdema has been long continued the limbs reach tremendous proportions. Some four years ago the writer had under his care at the Johns Hopkins Hospital a patient who reached the ward in a condition of very marked dyspnœa, with legs swollen to a diameter of 10½ inches and absolutely elephantoid in appearance (Fig. 123). The skin over the entire legs was covered with papillomatous outgrowths so suspicious that the diagnosis of elephantiasis vera was seriously entertained by some members of the hospital staff. The patient had been suffering from a severe aortic insufficiency for about a year, and for six months had been so orthopnœic that he had not been able to go to bed, but had rested sitting bolt upright in a chair and usually with feet down. There were tremendous crypt-like ulcers about 10 cm. (4 inches) in diameter in both legs, each covered with a deep layer of necrotic tissue. The patient was placed in bed with feet raised to the horizontal, and the wound dressed with a 1–10000 potassium permanganate solution and tr. digitalis m. xv administered every four hours. He was purged freely with Epsom salts. He improved so rapidly that within twelve hours the circumference of the legs had appreciably diminished and within a few weeks they were almost normal. The patient has remained quite well ever since and is at present managing a farm in western Maryland.

Unfortunately, œdema does not always disappear so satisfactorily. It has been shown that the œdema fluid is richer in salts than is the blood, and that resorption of the œdema is somewhat favored by a practically salt-free diet, such as one consisting of milk, sugar, eggs, meats, bread, sweet butter, and cereals prepared without the addition of salt. The diuretics, especially those of the caffein group, also favor resorption;[1] free-

[1] Fleisher, Hoyt, and Loeb have shown that the presence of calcium salts in infusion fluid diminishes the formation of œdema, but this observation had not yet received an application in therapeutics, and these observers have found that this does not apply to cardiac œdema.

purgation aids in removing fluid from the body, sometimes as much as 3000 c.c. per day, and digitalis does the same by increasing heart action. Besides this, the œdema can be combated by raising the legs to the horizontal or if possible a little above it, thereby increasing the drainage from them.

Drainage of Legs.—Sometimes also a light spiral elastic bandage of rubber dam may be applied to the legs, beginning at the feet and ascending to the groin, thereby assisting the drainage and replacing the lost elasticity of the skin. When these means are insufficient, several incisions may be made aseptically in the skin of the legs and drainage aided by applying Bier's suction cups or a large trocar with sides perforated (Curschmann's modification of Southey's tubes) may be inserted to bring about free mechanical drainage.

The best results are obtained by the following method, described by Romberg: Boil a long rubber tube fitted with a pinch-cock in physiological salt solution. After removing the point from the trocar, attach the rubber tube to the metal tube of the latter and place the lower end of the rubber tube in a basin of boiled water upon the floor next to the bed. In this way a siphon is established, helping the drainage of the fluid. The tube of the trocar should be kept in place in the leg by tying with a silk ligature, the ends of which are then inserted in a strip of adhesive placed loosely about the leg above the trocar. Romberg states that from 2 to 15 litres of fluid may be removed in 24 hours!

Fig. 124.—Curschmann's modification of the Southey tubes for draining œdema of the legs. (After Romberg.)

The œdema often involves the scrotum and penis until they are enlarged to two or three times the ordinary size, and phimosis or paraphimosis may cause considerable difficulty in micturition.

Ascites. — Ascites is common and may cause distress by pushing up the diaphragm as well as by impeding the abdominal circulation. It is often advisable to remove it mechanically by introducing a trocar in the midline midway between the umbilicus and the symphysis, under aseptic precautions (after first emptying the patient's bladder), and allowing the fluid to drain out. In so doing care must be taken to keep a many-tailed (Scultetus) bandage tight across the abdomen, readjusting it several times during the tapping for fear too much blood may enter the relaxed abdominal vessels when the pressure about them is diminished and shock result therefrom (see page 31).

Hydrothorax. -In cases with severely impaired compensation hydrothorax (usually right sided) is common. Starling and Leathes, Stengel, and Dutton Steele have demonstrated that its frequency upon the right side is due to the position of the great azygos vein, which drains the intercostal spaces and the pleuræ. One of the factors producing stasis in this vein is the fact that the latter enters obliquely into the superior vena cava (Fig. 5) and thus its mouth is readily closed off. Another is probably the fact, which the writer has noted, that the mouth of the vein is not as distensible as the walls of the vein above it, and hence imposes some obstruction to the blood flow. In all cases of heart failure in animals the azygos vein may be seen to be dilated above its entrance into the vena cava. The heart in hydrothorax is usually displaced to the left. The respiration shows

much embarrassment (1) from removal of a considerable part of the right lung, (2) from compression of the left lung by the displacement of the heart, (3) from embarrassment of the heart itself from the displacement, (4) from compression of the venæ cavæ especially during defecation and exertion. This may prevent inflow into the heart and cause sudden death. (Calvert.)

The fluid should always be removed promptly by paracentesis thoracis. This process is unfortunately not without danger, sudden death occasionally resulting in spite of the greatest care.

The writer has seen two deaths of this kind, and they occur with about the same frequency in the experience of most clinicians. A very valuable contribution to this field has been made by the studies of Capps and Lewis, showing that the visceral layer of the inflamed pleura is especially sensitive, and upon handling or touching it two reflex phenomena result, a vagal inhibition, sometimes so intense as to stop the heart, and a paralysis of the vasomotor centre which gives rise to a marked fall of blood-pressure. According to these observations it is therefore advisable to diminish vagal tone with a preliminary hypodermic injection of 0.5 to 1 mg. atropine (gr. $\frac{1}{12}$ to $\frac{1}{6}$) about 15 minutes before beginning the tapping, and to have at hand a hypodermic syringe loaded with 1:10000 adrenalin chloride solution to restore promptly the vasomotor tone in case of collapse.

RENAL COMPLICATIONS OF CARDIAC DISEASES.

RENAL CHANGES.

Albuminuria and diminished secretion of urine are invariable results of broken compensation. They may also occur after severe exertion, probably as the result of excessive pressure in the veins. The stasis in the vena cava and renal vein has been shown to give rise to albuminuria, and the slowing of the circulation through the kidney is sufficient to account for the diminution of the urine secreted. Such urine, though diminished in amount, is highly colored, normal or increased in specific gravity (1016 to 1026), and contains a normal concentration of NaCl and urea. The total excretion of the latter in 24 hours is, of course, decreased. As in the experimental stasis numerous finely granular and hyaline casts may be excreted.

The kidneys of such cases (Osler's arteriosclerotic kidneys) are usually of the large red or "beefy" type, with both cortex and medulla increased in size, the capsule adherent, and diffuse interstitial changes as well as some parenchymatous degeneration. The red color is probably due to the venous congestion.

It is of great importance to differentiate between a primary cardiac disease with secondary renal involvement and primary chronic nephritis with secondary arteriosclerosis, cardiac hypertrophy, and cardiac insufficiency. At an early stage of the disease a careful study of the chloride metabolism and its relation to urine concentration may be of great help.

In cardiac cases the power of excreting NaCl is, as a rule, not as much impaired as in renal cases. If 5 Gm. NaCl be added to the diet on one occasion there should be an abrupt rise in the NaCl content of the urine. If the kidney cells are damaged it will be gradually excreted during 2-4 days. However, it must be remembered that in many cases of primary cardiac origin the kidney cells may be impaired. The urinary findings, as shown in the cases cited on page 240 and page 335, may be practically identical, and hence all the features in etiology, history, and metabolism must be thoroughly considered before a decision is reached.

SENSORY SYMPTOMS ABOUT THE HEART

Palpitation.—Under normal conditions one is not conscious of sensory impressions from the region of the heart. Einthoven, Flohil, and Battaerd have shown, however, that an afferent impulse is transmitted up the vagi by each heart-beat, and there are probably similar impulses transmitted through the intercostal nerves from the parietal pleura, mediastinum, and chest wall, against which the heart is beating. Under normal conditions these sensations do not reach consciousness, but they occasionally do so when the general nervous sensibility is increased, as by coffee, tobacco, or hyperthyroidism, in neurasthenic and hysterical states, or when the beat of the heart is more forcible than usual. The distinct sensation caused by each beat of the heart is known as palpitation. It is frequently associated with cardiac weakness and irregularities, and has been thought by some writers to be clearly associated with extrasystoles. But while it is true that

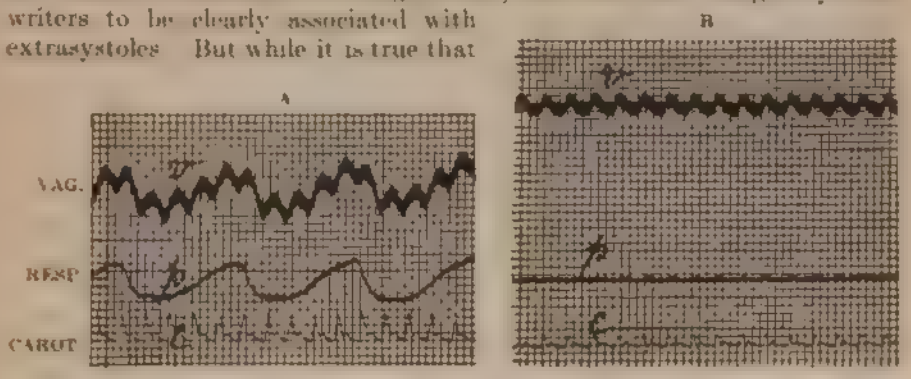

FIG. 125.—Electrical record of afferent impulses travelling up the vagi. After Einthoven, Flohil, and Battaerd. *VAG.* record of centripetal electrical wave in the vagus. *RESP.* respiration. *CAROT.* carotid tracing. A. In quiet breathing. B. In apnœa. The large electrical waves are due to respiration, the small ones to the cardiac contraction.

extrasystoles frequently give rise to palpitation and also that the patient can often distinguish between strong beats and weak beats, nevertheless there are many cases of extrasystoles without palpitation and of palpitation without extrasystoles. Hirschfelder has shown that palpitation may occur without any motor disturbance in the heart's action and without any change in the reflex response of the heart to various stimuli. Hewlett has found the *c* wave unusually large and sudden in a case of palpitation, and believes that the "earlier movements of the ventricle were exerted with unusual speed." However, this finding is not uniform in cases of palpitation, and, moreover, would not explain the occurrence of palpitation from weak extrasystoles in which these movements are executed slowly. Such changes in the venous pulse are often found in vigorously beating hearts without any palpitation whatever. Palpitation is therefore to be regarded as a purely sensory phenomenon, which, though it is frequently associated with cardiac disturbances, may occur quite independently.

The category of sensations in which palpitation should be placed and the path which the sensation traverses are not perfectly clear. It is evidently a pressure or touch sensation, perfectly distinct and limited to the period of systole. It is always sharply localized, usually referred to either the apex or the bifurcation of the trachea—the two sites at which

the heart exerts direct pressure or traction upon the surrounding structures. In this distinctness it differs entirely from other visceral sensations, which are less definite in time and in site, and also, as a rule, more nearly allied to pain sensation. It thus differs greatly from the pain sensations arising in and about the heart, and hence suggests that it should be placed in a different category. Whether the path of the sensation is up the vagi or through the thoracic nerves cannot at present be stated.[1]

Another point in favor of the view that palpitation is an extracardiac sensation is the fact that quite similar sensations may be felt in the abdominal aorta and radial arteries when there is visible pulsation (to-and-fro motion) of the latter with pressure and traction upon the skin and surrounding structures.

The continuance of the sensation is very wearing upon the patient, especially when the heart is irregular. Often he is able to distinguish between large and small beats, and is constantly reminded of the pathological condition and usually much worried about it. The sensation is not entirely dependent upon psychic phenomena; for in one case under the writer's observation (l.c.) it disappeared for five minutes, while the blood-pressure and pulse-rate rose after exercise when the patient was under examination, although he had his mind fixed upon the disturbance throughout the entire procedure.

Palpitation is frequently the result of tea or coffee drinking, smoking, digestive disturbances; it often occurs with various forms of cardiac diseases, but seems to have no relation to the latter. Relief is very difficult to obtain. Except for removing the causal factor, application of an ice-bag or a cold-water coil over the heart is about the best remedy. The bromides of potassium, ammonium, and strontium are of some value, as are also vibratory massage and the application of sinusoidal currents.

Precordial Pain. — Precordial pain is a less definite sensation than palpitation. It is continuous throughout the cardiac cycle, is less definitely localized, and more commonly associated with referred sensory disturbances resembling other types of visceral sensation. It seems to bear a somewhat closer relation to dilatation of the heart, and, as a rule, accompanies more severe organic diseases, being especially common in aortic and mitral lesions. There is sometimes, but not always, precordial tenderness.

The most severe form of precordial pain, angina pectoris (see page 285), in which there is, besides intense pain, a feeling as though the heart were held in a vise, seems to be associated with sclerosis of the coronary arteries, but a definite relation between this and other forms of precordial pain has not yet been established.

Frequently in heart diseases, and especially in cases of aneurism and angina, there is marked pain radiating down either or both arms. In fact these may be the first symptoms given by an aneurism. It is easily seen from the distribution of the cardiac nerves (page 14, Fig. 10) that a lesion in the vicinity of the sympathetic fibres might give sensations which, reaching one of the cervical spinal ganglia, would be referred to its peripheral distribution in the cutaneous region innervated by that segment, usually down the arm. Hence the frequency of these pains. Not only cardiac condition, but high blood-pressure in the aorta may cause this distress. It is difficult to relieve this symptom. If lowering of the blood-pressure with nitroglycerin fails to bring it about, codeine, 15 to 20 mg. (¼ to ⅓ gr.) should be tried, and, if that does not suffice, morphine may have to be resorted to, but should always be avoided as long as possible.

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[1] In this connection it would be interesting to note whether palpitation occurs in cases of transverse lesion of the cord in the lower cervical or upper thoracic region, or whether it can be brought on in such persons by large doses of coffee.

DIGESTIVE DISTURBANCES.

One of the first effects of weakening of the heart is engorgement of the veins of the portal system, and this in turn brings about a catarrhal condition in the mucosa, and especially the gastric mucosa, with consequent symptoms of indigestion. Fermentation frequently takes place, and the inflation of the stomach with gas, displacing the diaphragm upwards and shifting the heart more towards the horizontal, tends to increase its embarrassment. Overloading the stomach, the transdiaphragmatic neighbor of the heart, should therefore always be avoided; and the patient will be saved much suffering if he is kept on a light, easily digestible diet, consisting largely of eggs, milk, and carbohydrates, just enough in quantity to keep him from losing weight. Friedrich Müller has shown a diminished power of absorption of fats in heart disease. Perhaps this may be due to the fact that the high venous pressure prevents the thoracic duct from emptying itself properly, or perhaps because, as H. M. Evans has shown, a high portal pressure causes the lymphatics of the intestines to collapse.

On the other hand, meats and other foods containing purin bodies in large quantities (sweetbreads, lungs, liver, etc., also coffee and tea, and alcohol in all forms) do distinct harm by raising the blood-pressure and by increasing the viscosity of the blood (page 39).

The engorgement of the gastric and œsophageal veins sometimes leads to exudation of blood into the stomach and to vomiting of blood.

Abdominal Pain from Distended Liver. One of the commonest symptoms of failing compensation is very intense abdominal pain felt over the region of the liver. This organ may become much distended, and, as shown by Salaman, may be expanded until its blood content is several degrees above the normal. Under this expansion there is marked tension upon the capsule of the liver (Glisson's capsule) which, in turn, gives rise to pain. This symptom is really so clearly bound up with the failure of compensation itself that it subsides with resumption of the latter, or after some time the capsule of the liver will have become sufficiently stretched and it will then cease to be painful.

Abdominal pain also results from arteriosclerosis of the gastric and mesenteric arteries, from vascular crises as well as from abnormally great pulsation of the abdominal aorta.

Catarrhal Jaundice. Like the gastric mucosa, the bile passages undergo catarrhal inflammation from the venous engorgement, and a definite catarrhal jaundice may accompany the failure of compensation. Usually, the jaundice is mild and barely perceptible, the color being sallow and icteroid rather than icteric. The presence of this slight icteric hue in a patient with heart disease should always lead to the suspicion of broken compensation or tricuspid insufficiency, and is always a sign of danger.

PSYCHIC DISTURBANCES.

An anxious expression is so commonly manifested by patients suffering from heart disease that a certain type is spoken of as "the cardiac facies." This facies is difficult to describe, but may be said to be character-

ized by bright watery, somewhat staring eyes, wide palpebral slits (without definite exophthalmus or other signs of Basedow's disease), rather tensely held mouth, and the rest of the face a little sunken, though not to the degree present in the "abdominal facies." Many cardiac cases, perhaps from the difficulty which they are constantly experiencing in getting their breath, feel irritable and peevish to a considerable degree, and not infrequently the onset or increase of peevishness is an early sign that the cardiac condition has become worse.

DELUSIONS.

Occasionally, especially in patients with irregular heart action, definite psychoses set in. These are especially common during the night and early morning, disappearing again during the waking hours.

The patient usually awakens from his sleep unable to recognize the place where he is, which he usually locates somewhere else, and then regards the doctors, nurses, and attendants as inhabitants of the more familiar scenes often mistakenly recognizing them as people of his acquaintance in those places. He usually regards his confinement in bed as a sign of some attempt upon his life, and the administration of medicine as a certain attempt to poison him. Of this fact he is always certain, although he may admit that there is some doubt in his own mind as to the correctness of some of his other ideas. For example, one delirious patient under the writer's care as house physician mistook him for an old friend from home and said, "He liked ——— and had great confidence in him but he could not see why ——— did want to poison him." But he would recognize no other possible motive.

Occasionally when daylight comes or some one familiar object appears, the patient suddenly recognizes his surroundings, wakes up as from a dream, and may even explain exactly the nature of and reasons for his delusions. Under the influences of these delusions, patients are often very hard to manage, but their attitude is more commonly a defensive than an offensive one, resisting confinement and treatment, and attempting to leave the ward or room peaceably, rather than showing maniacal pugnaciousness primarily. They can usually be persuaded by gentle means to remain where they are for a time, especially as their minds are almost always confused; they realize that they are not perfectly well, and the nurse or physician can lead the argument along its logical conclusions to a reason why they should return to bed and to rest for the time being. After some minutes' argument of this kind the patient can usually be given a hypodermic of morphine and gotten back to bed with much less injury to himself than if forcible means were attempted. He can then usually be kept in bed by an attendant constantly present.

The reason for these delusions is not very certain, but in some cases they may be regarded as "waking dreams" not very different from those of somnambulists, and perhaps like the night terrors of children with adenoids. They may be asphyxial in origin, associated more or less with cerebral arteriosclerosis and cerebral anæmia, of which perhaps the frequent high blood-pressure may be another expression. This delusional insanity is a bad omen, and its onset often precedes the fatal outcome by only a few days or weeks. Duroziez and H. O. Hall have called attention to the fact that in some cases these delusions may be due solely to the digitalis and may disappear entirely when the drug is discontinued.

HALLUCINATIONS.

Definite hallucinations of sight and hearing are also not uncommon.

Henry Head has observed that these are especially common in aortic disease about the time of twilight, and are usually quite simple in character, the auditory hallucinations

consisting in simple rhythmic sounds (associated with the heart-beats?), such as of knocking or of bells tolling, the visual hallucinations usually taking the form of the face of a man or woman seen stationary at the foot of the bed or slowly stalking across the room. The face is ashy white, the eyes black and staring, and the contour invariably indefinite and surrounded by a mass of wavy black hair. If the body is seen at all, it is poorly outlined as though draped in a black gown. Head found this hallucination quite constant and seen by many patients, though they as a rule, recognized the hallucinatory character and spoke of it only after the physician had gained their confidence. The writer has also elicited the same answers from a number of patients, after prefacing the question by a statement that visual hallucinations were not uncommon in their disease and were to be regarded merely as troublesome but not significant features of the disease itself. All who gave positive answers accurately described the hallucinatory vision as above.

Head states that highly colored and rapidly moving visions do not occur frequently in heart cases, but the writer has seen one very marked exception to this rule.

This was in the case quoted on page 508—a young railroad engineer, 23 years old, of temperate habits and excellent family history, who had a very adherent pericardium. For several years, especially when his cardiac condition became worse, he suffered from seeing a few feet before him swarms of large animals, lions, tigers, etc., all highly colored leaping rapidly about. He recognized these as hallucinations at the time, but stated that the sight irritated him so that he lost his self-control, and he begged to be placed in solitary confinement for a few days until the hallucinations passed off. He was then once more a perfectly rational being.

Like the delusions, these cardiac hallucinations are probably due either to anæmia or venous stasis in brain, but especially in the special centres, or in the retina, middle ear, visual or auditory centres, giving rise to rudimentary sensations which the mind translates or distorts into the above-mentioned pictures.

Syncopal attacks also occur in some forms of heart disease as a result of cerebral anæmia and will be discussed in detail in Part III, Chapter XI, under the head of Adams-Stokes disease. The feeling of faintness and weariness unaccompanied by syncope will be discussed under "cardiac neuroses," etc. (Part IV, Chapter III).

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IV.

GENERAL PRINCIPLES OF TREATMENT OF FAILURE OF THE HEART.

The best index of the treatment of the patient is his own condition, sensations, and general appearance. Physical examinations, determination of blood-pressure and pulse-rate, as well as of increase in the product of pulse-pressure by pulse-rate (velocity coefficient), venous tracings, and gas analysis aid in the interpretations of the condition, and particularly in discovering where the fault in the mechanism of the circulation lies; but the changes of conditions themselves are often very subtle and manifest themselves in the general condition of the patient before they can be detected on examination.

QUIET.

The most important element in the treatment of cardiac failure is *rest* as complete as possible. In all cases of heart failure or disease in the heart the patient should be confined to bed, if necessary propped up with pillows, and should be kept there until the acute symptoms have subsided and have remained quiescent for several days.

As Morton Prince has shown, *mental excitement* and *worry* are important factors in bringing about acute dilatation of the heart; but they are usually contributing factors rather than sole causes.

The effect of mental activity upon the circulation is to bring about vasoconstriction, of both extremities and viscera, a slight rise of blood-pressure, and increase in the pulse-rate; all of which taken together considerably increase the total work of the heart (*i.e.*, roughly speaking, the product of maximal pressure by pulse-rate).

A good night's *sleep* is often the best remedy for the patient with a weak heart, and almost any method by which it may be procured may prove a good therapeutic procedure. Small doses of bromides, if necessary aided by a little trional, veronal, or other hypnotic, often suffice for this purpose and allow the heart a few hours respite in which the other therapeutic measures may have opportunity to act.

However, it must be borne in mind that in persons who are much worried, *mental rest and absence of distractions or occupation are not necessarily synonymous*. Indeed, the removal of other subjects for thought may serve only to centre the patient's mind upon himself and his ailments and may increase rather than diminish the nervous strain. This should be carefully guarded against. The daily routine should therefore be accommodated to both the general condition and the temperament of the patient. If possible absolute rest and isolation should be secured for the worst cases of heart failure, but even for these patients a few minutes' conversation with a cheerful friend, whose demeanor is quiet and soothing, may be of actual benefit. Reading should not be allowed to

patients in the worst stages, but a little reading of the lightest and least exciting sort may otherwise be allowed.

Rest, Distraction, and Spa Treatment.—As Mackenzie states, it is chiefly due to the element of mental distraction combined with the judicious supervision of a physician and the favorable climatic conditions, which make the Spa treatments of cardiac disease so successful; although, as he states, each Spa physician has evolved some method of treatment which he regards as of special benefit, when the actual benefit has been due to the air and restfulness itself. Nevertheless, it must be confessed that the treatments at Nauheim by the late August Schott (page 194) have been of great benefit, and being founded upon sound physiological doctrines have been applicable elsewhere as well. The physician must always realize that, however little there may be in the Spa treatments *per se*, the combination of the mental rest and change of air with the baths and dietetic treatment is one which the patient whose condition warrants a trip, should not forego.

Rest in Bed.—The bed should not be so high above the floor as to make it hard to get in and out; it should if possible have a metal frame and a good rather firm mattress. It should be provided with a good back rest ready for use in case the patient finds it more comfortable, and plenty of pillows should be available.

In dealing with cases of mild cardiac failure it may be impossible in private practice to compel the patients to remain in bed all the time, and then it may suffice to insist upon their lying down for several hours a day without absolutely remaining in bed. Under these circumstances the physician must insist that the patient remain quiet all day upon a sofa or in a wheel chair with legs raised. A short period of such absolute rest is better than a much longer period of relative invalidism, for it enables the dilated heart to bail itself out, to regain its former dimensions and tonicity, and permits the heart-rate to return to normal. It is important that the patient should remain horizontal rather than in sitting or in standing posture, since the latter tends to slow the circulation (cf. Erlanger and Hooker, quoted on page 26). The rest should continue until all symptoms have subsided, until cardiac distress, pain, and palpitation have disappeared, and respiration has again returned to normal. If possible the subsidence of tachycardia or irregularity of the pulse should be awaited, but these may persist for some time even in spite of the improvement in the patient's general condition, and may have to be disregarded. After the symptoms have subsided (in severe cases after the symptoms have remained quiescent for a few days), the patient may be allowed to get out of bed and sit up in an arm chair or wheel chair for a little while. At first this period should be very short, to avoid exhaustion, but it may be gradually increased and he may soon be allowed to walk. (For exercises to be taken by patient with cardiac disease see page 199.)

COLD APPLICATIONS OVER THE HEART.

The application of cold to the precordium is of value both for the cardiac symptoms (palpitation and pain) on the one hand, and for diminution of the heart-rate on the other. This may be carried out by the applica-

tion of a simple ice-bag (especially containing a mixture of ice and salt) which may be kept in close application to the skin by tying it around the chest and shoulders with a strong elastic four-tailed bandage. The ice-bag should be changed every hour or two in order to keep up an intense cooling.[1]

In hospital use or in well-supplied houses the use of the cardiac tube is most satisfactory. This consists of a coil of thin-walled rubber or aluminum tube applied over a wet compress to the precordium. A stream of cold water from a cooler is kept flowing slowly through the tube. The cooling of the skin thus obtained is excellent and without any discomfort to the patient. Its effects have been tested both clinically and experimentally by Winternitz and da Silva.

These observers found that the application of cold to the precordium brought about in dogs a cooling of both the anterior and posterior surfaces of the pericardium, amounting to 1° 5°, and was accompanied by a slowing of the pulse and rise of blood-pressure from 120 to 190 mm. Hg. In man the pulse-rate did not begin to fall for fifteen minutes after the application, and reached its height within an hour, lasting in turn about an hour after removal of the cold. In normal individuals they found the pulse-rate falling from 72 to 61, 68 to 52, 78 to 68; in other cases, chlorosis 84 to 72, pericarditis 81 to 78, mitral stenosis 60 to 40. Simultaneously the blood-pressure rises and the pulse increases in volume. There is evidently both a reflex vasoconstriction from stimulation of the vasomotor centre and a reflex stimulation of the vagus. Besides this, da Silva thinks that there is a direct stimulation of the heart muscle. It will be noted that these effects are exactly those brought about by digitalis, and hence enthusiastic hydrotherapists are in the habit of speaking of the ice-bag as "physiological digitalis."

Its use is attended with less danger, but in cases of extreme fibrous or fatty degeneration of the heart, cyanosis and collapse occasionally occur. Hence it should be applied very mildly in cases where these conditions are suspected.

There can be no doubt that the ice application is not as efficient as the use of digitalis in slowing and strengthening the heart; but when the two are vigorously used at the same time they may greatly reinforce one another, and the vigorous use of a good ice-bag may enable satisfactory effects to be obtained with smaller doses of digitalis than would otherwise suffice.

VENESECTION.

When the patient is in very bad condition, deeply cyanotic, and restless or nervous, and the area of cardiac dulness is increased to the right, a free venesection will often bring the greatest relief.[2]

Technic of Venesection. – Venesection is best performed in the following manner: The skin over the flexor surface of the elbow-joint is scrubbed with green soap and washed with warm water, then with alcohol, and lastly with 1-2000 bichloride solution. An elastic or gauze bandage is tied about the upper arm tightly enough to cause the veins to stand out but not to obliterate the pulse at the wrist. The largest vein visible (usually the median cephalic) is selected and a small slit in the skin just alongside of (not over) the vein is made with a curved bistoury, which is then pushed in through the slit in the skin and under the vein. It is then twisted so that the edge is turned upward towards the skin against the vein and the vein cut through without again piercing the skin. A very free flow of blood is obtained, especially by keeping the arm dependent and if the patient is made to clench and open his hands rapidly. From 300 to 1200 c.c. (12 ounces to 2½ pints) can thus be removed in less than twenty minutes, usually with great relief to the patient. Breathing

[1] When a mixture of ice and salt is used it is possible to actually freeze the skin, an accident which must be carefully avoided.

[2] The hæmoglobin should always be tested before performing a venesection; and it should not, as a rule, be performed if the hæmoglobin is below 70 per cent.

becomes easier, the head clearer, and the general condition better, but the crucial point is reached when the color changes and the cyanosis gives way to a healthy rosy color in the lips and elsewhere. This indicates that the overstrained heart has been unburdened, and the bleeding need not be pushed much further. Indeed it should not be, for to cause an anæmia is dangerous. All that is desired is to relieve the distention of the right heart.

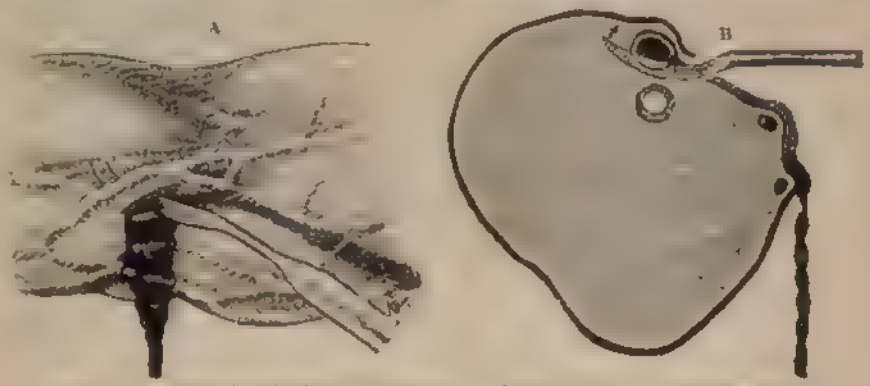

FIG. 126.—Insertion of the knife in venesection. A. Lateral view. B. Cross-section of arm.

Effect of Venesection on the Circulation.—The value of venesection can often be seen in experiments upon animals. It is not at all uncommon to find a heart failing and an auricle already paralyzed from overdistention, in which a free venesection gives immediate relief, and the auricle as well as the ventricle resumes forcible contractions. The effect of this procedure upon the blood-pressure is variable and depends to a certain extent upon the phenomena present before the venesection.

Before Venesection		After Venesection
Blood-pressure	Condition	
1. Normal or elevated	Heart distended but circulation still sufficient	Fall of blood-pressure from emptying of vascular system and diminished viscosity of blood.[1]
2. High	Circulation slowed. Vasoconstriction through stimulation of medulla by excess of CO_2 in the blood	Fall of blood-pressure, occasionally compensated by increased force of heart-beat and diminished viscosity of blood.
3. Low or normal	Circulation slowed, heart failing. Unable to keep up circulation through medulla in spite of vasoconstriction	Blood-pressure rises on account of marked increase in force of heart-beat in spite of emptying of vascular system and of relaxation of peripheral vessels.

Quite independently of these changes the right border of cardiac dulness recedes one or more centimetres toward the sternum, the venous pressure should fall, and the general condition should improve (cf. Fig. 127 and case on page 239).

Contraindications to Venesection.—However, it must be borne in mind that venesection can do harm as well as good. Cushing has shown that in conditions with increased intracranial tension, among them apoplexy, the

[1] Heubner has shown that two-thirds of the viscosity of the blood is due to the corpuscles, hence venesection cannot fail to reduce the viscosity.

high blood-pressure is a phenomenon of physiological compensation, which is necessary in order to maintain the circulation through the medulla. In conditions with long-continued high blood-pressure, especially chronic nephritis, this may also be the case. In these conditions venesection with a view to lowering the arterial pressure is contraindicated; but in these, as in other conditions, it is still the procedure of choice to relieve pulmonary œdema or acute dilatation of the right heart. The venesection should be carried only to the point of relieving the venous stasis, not to that of lowering the arterial pressure.

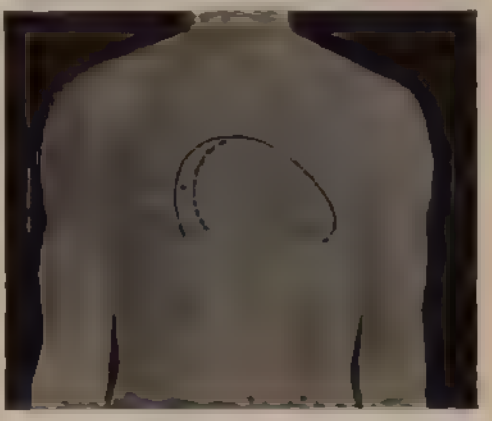

FIG. 127.—Effect of venesection on the cardiac outline, showing diminution in size of right heart. Case of C. C. Solid line indicates cardiac outline before venesection, broken line after venesection.

DIET.

Rest for the gastro-intestinal tract is quite as important for the heart as is rest for the muscles. Erlanger and Hooker have shown that "an increase in pulse-pressure becomes manifest within a few minutes after the beginning of the meal, reaches its maximum within one or two hours, and, as a rule, declines somewhat more slowly. It seems to pass off within one or two hours after the maximum has been reached. The pulse-rate is always distinctly increased with the ingestion of meals. . . . . The product P. P. × P. R., representing the velocity, follows the curve of the pulse-pressure," hence the velocity of flow and the work of the heart are increased. Accordingly, the diet should be light, just enough to keep the patient nourished without ever giving him a sense of fulness or to allow gas to form in the stomach and intestines. Distention of the stomach pushes up the diaphragm and causes the heart to lie more transversely in the thorax, embarrassing its action, causing a diminution in the systolic output and an increase in the pulse-rate. Not infrequently this is also associated with onset of precordial pain and constriction. Accordingly a very light diet is necessary for the patient suffering from heart failure. The lacto-cereal diet is the best, consisting mainly of milk, eggs, custards, junket, toast, zwieback, crackers. The numerous prepared cereal foods,

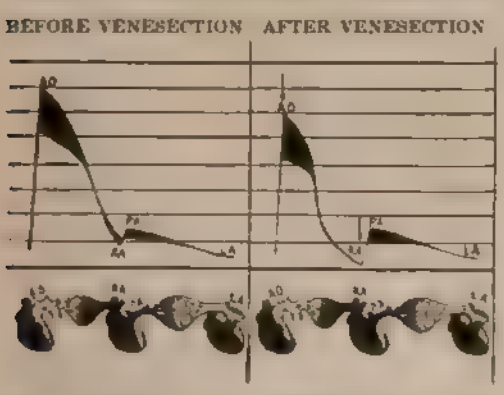

FIG. 128.—Typical effect of venesection upon the circulation. Arrows indicate change in blood-pressure.

which consist of partially toasted flakes of wheat, corn, or rice, are particularly good, since much nourishment may be given in small bulk and in a form which does not tend to form thick, impenetrable, doughy masses. Besides they contain the bran as well as the starch, and hence, by leaving considerable fecal residue, tend to keep the bowels moving. Meat should be given sparingly, partly because the purin bodies (xanthin, hypoxanthin) tend to raise the blood-pressure and increase the work of the heart, and more particularly because the meat fibres are relatively slow in digestion. For this reason it is better to take the proteid food in the forms mentioned above. Finely hashed Hamburg steak, lamb chops, or chicken are the best forms of meat.

Liquid and Salt.—Liquids should be limited to 1500 c.c. (three pints) a day in cases where œdema is present, since an excess of liquid ingested causes further accumulation of œdema as well as bringing on a slight overfilling of the blood-vessels, and thereby increasing the work of the heart.

Salt should also be withheld from the food as far as possible, since Widal and Javal, Strauss and Richter have shown that it is a contributing factor in the production of œdema, and Barié reports good results from the diminution of NaCl in the diet in diseases of the circulation.

Barié recommends the following articles as a basis for a diet low in sodium chloride. Type I.—Unsalted bread 500 Gm. (18 oz.), raw meat 400 Gm. (14 oz.), butter 80 Gm. (2½ oz.), sugar 100 Gm. (3½ oz.). Type II.—Potatoes 1000 Gm. (32 oz.), raw meat 400 Gm. (14 oz.), butter 80 Gm. (2½ oz.), sugar 150 Gm. (5 oz.).

Sample Diet.—An excellent diet for severe heart cases, which may at least serve as a basis for other variations, is the following, slightly modified from that used for cardiac cases in the wards of the Johns Hopkins Hospital:

8 A.M.	Cereal, soft egg, toast, coffee 200 Gm. (vi oz.).
10 A.M.	Milk 200 c.c. (vi oz.), soft egg, crackers.
Dinner (noon)	Soup, chicken, potatoes.
4 P.M.	Milk 200 c.c. (vi oz.).
Supper, 6 P.M.	Milk 200 c.c. (vi oz.), soft egg, crackers, prunes.[1]
9 P.M.	Milk 200 c.c. (vi oz.), bread.

Limited Milk Diet.—In cases of broken compensation with extreme œdema, great success has sometimes been attained by limiting the diet to 600 to 800 c.c. of milk in 24 hours (Carell, Hoffmann, Jacob and Hirschfeld), even in cases in which all other therapeutic measures have failed. Professor Barker has occasionally obtained excellent results by increasing the proteid intake upon this diet through the addition of nutrose to the milk. However, striking results with this method are by no means the rule, and it is to be used with caution.

Alcohol.—A very little alcohol, either as wine, or as brandy or whiskey, may be allowed to persons accustomed to its use. Beer is less advisable,

[1] It is important to avoid giving stewed fruits which contain much acid, such as peaches and apricots, along with the milk, as the digestion of patients with broken compensation is very easily disturbed, and an attack of vomiting places a considerable strain on the heart.

since it carries with it large quantities of liquid and often disturbs the digestion as well, whereas, wine, whiskey, or brandy in small quantities improves it. Against this is balanced the deleterious effect of alcohol upon the heart muscle. Large quantities tend to produce fatty degeneration of the latter. Whether small quantities have any such effect in the individual case is uncertain, but it must be borne in mind that the injured organ is much more susceptible to deleterious influences than is the healthy organ. It is a safe rule that, in persons not already addicted to its use, brandy or whiskey be given only in doses which serve as carminatives, and not in doses intended for stimulation. Even the psychic effect may often be secured as well by small doses as by large ones. One point in favor of alcohol in man as against animal experimentation lies in the fact that in such persons it greatly increases the sense of well being and removes psychic depression and worry. The latter may be especially straining upon the heart, and hence every effort should be made use of to ward it off, especially during certain crises, but it should be borne in mind that the patient may easily become dependent upon the drink to arouse his spirits and in this state more harm than good is done. The greatest judgment should be used in the administration of alcohol even in small quantities, and it should even then be reserved for crises when the stimulation of every fibre is all-important. On the other hand, alcohol should never be withdrawn suddenly from persons addicted to its use, since this procedure often precipitates an attack of delirium tremens, but moderate doses (whiskey 15 c.c. or ½ oz. every four hours) should be given.

Tea and Coffee.—Whether tea and coffee should be given depends largely upon the patient. In some persons these cause marked general nervousness, sleeplessness, tremor, and even palpitation and irregularity; others have established a tolerance such that no effect at all is produced. The caffein itself is an excellent cardiac tonic of the digitalis order, and where its effects on the nervous system are not manifest it may prove an excellent adjuvant to the treatment. (A cup of coffee or of strong tea contains about 0.1 0.2 Gm., 1½ to 3 gr.; the pharmacological dose of pure caffein being 0.05 to 0.25 Gm.) As a rule it is safer to remove them from diet, but in this as in all other rules individual exceptions can be made.

Tobacco should not be used under any circumstances. Besides the nervous symptoms, it produces vasoconstriction, and often irregularities, palpitation, and even precordial pain. Hence it is particularly to be avoided in cases of cardiac disease.

PURGATION.

In patients with cardiac disease, and especially in those with broken compensation, the question of purgation assumes unusual importance. In these patients purgation seems to have a threefold beneficial action: first, by eliminating the products of waste and putrefaction, to which they are particularly sensitive; secondly, by relieving the distention of the bowels from gas which tends to push up the diaphragm and to embarrass the heart by placing it in a more transverse position; and thirdly, by removing fluid from the body through the bowels. This last effect is probably of con-

siderable importance, since Askanasy, Kast, and others have shown that broken compensation is accompanied by hydræmic plethora. Hydræmic plethora causes a rise in venous pressure and a dilatation of the heart (Roy and Adami, Cameron), thus embarrassing the circulation. Moreover, in broken systemic compensation the venous stasis also affects the kidneys and diminishes the excretion of fluid, so that the bowel becomes an important accessory channel of elimination. It is therefore the hydragogue purgatives which are indicated in cardiac failure and not merely the purgatives which increase peristalsis.

In most cases the best method of procedure is to start movement of the bowels with calomel in either large single doses (0.3–0.6 Gm., grs. v–x) or in small divided doses (.006 Gm., gr. $\frac{1}{10}$ half-hourly). The dose of calomel should always be accompanied by a small dose of bicarbonate of soda (0.3–0.6 Gm., gr. v–x) to avoid disturbing the digestion. Still more certain purgation is obtained by giving a single dose of calomel and rhubarb in equal quantities (0.3 Gm., gr. vi), given at night. In all cases the calomel should be followed by a saline purgative the next morning. Epsom salt or some aperient water is preferable to Seidlitz powders or effervescent citrate of magnesia, partly because of the action of the organic acids upon the residium of calomel, but chiefly because the carbonic acid in the drug distends the bowels and pushes up the diaphragm, thus embarrassing the action of the heart. However, Epsom salts and aperient waters sometimes cause nausea, and in such cases the advantages gained from the mildness of the Seidlitz powder may outweigh its deleterious effects.

After constipation has been overcome purgation with salines should be continued vigorously until the œdema has completely disappeared. Just how vigorously this purgation should be maintained is a matter of some dispute. Some clinicians, who regard presence of fluid as the most deleterious factor, believe that the best results are obtained with ten to fifteen fluid stools in twenty-four hours, with the elimination of two or three litres by the bowel. Most observers, however, believe that the beneficial advantages of such extreme purgation are more than counterbalanced by the strain which they place upon the patient, not only by disturbing his rest, but also by causing a considerable rise of both arterial and venous pressure with each movement of the bowels. Indeed, each effort at stool constitutes a typical Valsalva's experiment, which, as has been seen (Fig. 116, p. 132), is accompanied by tremendous rises in blood-pressure and in weakened hearts by acute dilatation.

Mr. W. E. Dandy has shown that the rise of arterial pressure during the act of defecation is from 30 to 50 mm. Hg, and Mr. C. C. Cody has found a corresponding rise in the venous pressure. These observations are supported by the fact that sudden death at stool is by no means uncommon in cases of cardiac disease, especially in cases of aortic insufficiency, and occurs even when the movements have been kept soft by daily purgation with salts.

In this, as in most other therapeutic procedures, extreme measures are to be avoided and treatment should be directed to secure a few easy bowel movements without too much disturbance to the patient. In many cases one or two compound cathartic pills (colocynth, jalap, gamboge, and

calomel) at night and a dose of Epsom salts or aperient water in the morning maintain just the correct number and quality of stools. Compound jalap or compound licorice powders are also useful from time to time. In stubborn cases elaterium or a drop of croton oil may be resorted to, but should be used with extreme caution.

On the other hand, cascara, aloes, strychnine, belladonna, castor oil, phenolphthalein, and the other purgatives which purge by increasing peristalsis, are of less value in the stage of broken compensation, since they do not deplete the portal system nor relieve the hydræmia, though they are satisfactory enough when compensation has been reëstablished.

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V.

THE EFFECTS OF DRUGS IN CARDIAC DISEASE.

It does not lie within the scope of this work to enter into a detailed discussion of the pharmacology of the drugs used. The reader is referred to the text-books upon this subject, especially—

Cushny, A. R.: A Text-book of Pharmacology and Therapeutics, Philadelphia and New York

Solmann, Torald. Text-book of Pharmacology

Hatcher, R. A., and Wilbert, M. The Pharmacopœia and the Physician, Chicago, 1907 (published by the American Medical Association)

Heinz, R. Handbuch der experimentellen Pathologie und Pharmacologie, Jena, 1905

However, since it is frequently inconvenient to refer to such books, a brief summary of the action of each drug will be given with especial regard to its clinical application

The drugs used in the treatment of cardiac disease may be of value through their action on the following systems:

I. Upon the Heart Muscle—digitalis, strophanthus, strychnine, squills, caffeine.

II Upon the Peripheral Vessels—*constrictors:* camphor, strychnine, adrenalin, ergot, digitalis, nicotine (tobacco), caffeine; *dilators* amyl nitrite, nitroglycerin, sodium nitrite, erythrol tetranitrate.

1 Acting upon the Cardiac Nerves.

A *Slowing the heart through stimulation of the vagus:* aconite, digitalis, strophanthus, sometimes strychnine and caffeine, nicotine, veratrum viride, muscarin, very large doses of potassium salts, bile salts, blood in jaundice

B *Increasing the heart-rate through paralyzing the vagi:* atropine, cocaine, amyl nitrite and other nitrites

C *Increasing rate through stimulation of accelerators:* adrenalin, amyl nitrite, and other nitrites

D *Paralyzing accelerators:* apocodein.

2 Diminishing Venous Pressure and Stasis by Depleting Portal System: purgative series, especially calomel, the saline and the vegetable purgatives.

3 Drugs which Increase the Tonicity of the Cardiac Muscle in pharmacological doses digitalis, strophanthus, strychnine, amyl nitrite, nitroglycerin, calcium chloride (transitory effect).

4 Drugs which Decrease Tonicity potassium salts, chloroform, formic acid, salt infusion ether, adrenalin.

Tonicity is practically unaffected by small doses of aconite, though slightly diminished by larger ones.

DIGITALIS.

Foremost among the drugs used in treatment of circulatory diseases are the preparations of digitalis, introduced into medical practice by Withering in 1785. He says of it: "In the year 1775 my opinion was asked concerning a family receipt for the cure of the dropsy. I was told that it had long been kept a secret by an old woman in Shropshire, who had sometimes made cures where the more regular practitioners had failed. . . . The medicine was composed of twenty or more different herbs, but it was not very difficult for one conversant in these subjects to perceive that

the active herb could be no other than the foxglove. . . . I soon found the foxglove to be a very powerful diuretic . . I use it in ascites, anasarca, and hydrops pectoris." He then cites the results obtained in the treatment of over 100 cases, many of which would be worthy of modern therapeutics.

Drugs of Digitalis Series.[1]—Digitalis, strophanthus, apocynum, convallaria majalis, squill (scilla), erythrophlæum, helleborein, antiarin (antiaris toxicara).

Digitalis consists of the dried leaves of *Digitalis purpurea* collected from the flower at the commencement of the second year's growth. It should not be kept more than one year.[2] Average dose pulv. digitalis = 0.05 Gm. (1 gram).

Preparations.	Dose	
	Gram	English
Fluid extractum digitalis	0.05	♏ 1
Extractum digitalis	0.01	gr. 1/5
Infusum digitalis (1.5% digitalis + 10% alcohol + 15% cinnamon water) .	8.00	℥ ii
Tinctura digitalis . (10% of crude digitalis in dil. alcohol)	1.00	♏ xv

A very satisfactory form for administering digitalis and a purgative at once is Addison's (or Niemayer's) pill, made up according to the following prescription:

Pulvis digitalis } aa	0.6	gr. x
Pulvis scillæ }		
Hydrarg. chlorid. mit.	0.08	gr. 1 1/4

M. fiat in pil. x seu capsulas x.

Sig. One pill every three hours.

The calomel may be increased to gr. x, or may be replaced by blue-mass (massa hydrarg.) or gray powder (hydrargyrum cum creta) in capsules.

The efficacy of Addison's pill depends upon the care taken to secure an active preparation of digitalis in making it. Moreover, its action may be uncertain, owing to the fact that a certain amount of digitalis is eliminated with the stool without having been absorbed.

Derivatives of Digitalis.—*Digitoxin*—the most active substance derived from digitalis, producing all the digitalis effects, soluble in alcohol, insoluble in water, except in the presence of digitonin. Prepared in soluble form with digitonin under the trade name "Digalen" (Cloetta). "Digalen," dose 1 c.c.

Digitalin (digitalinum verum Kiliani)—a white amorphous glucoside, less toxic than digitoxin but otherwise resembling it in physical properties and pharmacological action. Dose 2.6 mg. (gr. $\frac{1}{25}$ $\frac{1}{15}$).

Roughly, digitoxin is six times more potent than an equal weight of digitalinum verum (Fraenkel).

Digitalin "German"—amorphous powder, soluble in water and alcohol; a mixture of pure digitalin, digitalein, and digitonin. Dose 2.6 mg. (gr. $\frac{1}{25}$ $\frac{1}{60}$).

Digitalein and *digitonin* are other somewhat similar substances which have no pharmacological action.

Strophanthus—the ripe seeds of Strophanthus Kombé. Tinctura strophanthi, 10 per cent. of the drug in 65 per cent. alcohol. Dose 0.5 c.c. (♏viii).

[1] A very full discussion of these drugs is given in Cushny's article.

[2] It is most difficult to obtain a preparation of digitalis leaves of which one can be certain, and upon this alone the result of the whole treatment depends. Owing to the variations in leaves it is best to obtain preparations from manufacturing chemists who have standardized them from physiological effects upon frogs or mammals. (The term "frog unit," "Froscheinheit," refers to the amount of drug which will kill an average frog, leaving the heart in systole, cf. also Sowton and especially Edmunds and Hale.) Edmunds and Hale have shown that in frogs the drug acts chiefly on the heart, in mammals largely on the nervous system.

Derivative and Active Principle.—*Strophanthin*—a white crystalline glucoside of constant composition and action, soluble but undergoing decomposition in water. Hence best prescribed in dilute alcohol:

Strophanthin	0.01	gr. 1/5
Alcohol dil	1.50	℥ v

Sig. Teaspoonful p. c. in half glass of water.

R. A. Hatcher has shown recently that the absorption of strophanthus and strophanthin from the gastro-intestinal tract is very uncertain, and that it is liable to set in suddenly after tremendous doses have been given without effect. Hence these drugs should be administered intravenously or intramuscularly.

For intravenous or intramuscular injections strophanthin (amorphous) is put up in small sterile phials (Boehringer & Co.). It is very useful for the physician to carry a few of these in his emergency case.

Strophanthin (Thoma) is said to be crystalline and is a more stable and more uniform preparation. It acts in doses of ½ to ¼ mg (gr. $\frac{1}{120}$ to $\frac{1}{60}$).

EFFECT OF DIGITALIS ON THE NORMAL HEART.

Fraenkel and Schwartz and also Cloetta have shown that in therapeutic doses digitalis has no effect upon the normal heart, either in affecting the strength of the beat or in bringing about hypertrophy. Neither has it any effect upon the perfectly compensated, undilated heart with a valvular lesion. Its chief effects are seen in dilated hearts whose myocardium still retains some reserve power. In the severest stages of cardiosclerosis and fatty degeneration it may stimulate the fibres to the limit of their power, and thus do actual harm, and even hasten the end.

ACTION OF DIGITALIS.

Digitalis has the following actions: (1) it stimulates the vagus, both centrally and peripherally, brings about slowing of the heart, and diminishes conductivity; (2) it increases the irritability, force of contraction, and tonicity of the cardiac muscle in both auricles and ventricles, slightly diminishing conductivity by direct action upon the cardiac muscle even in atropinized hearts; (3) it causes the peripheral blood-vessels to constrict, thereby raising the blood-pressure; (4) a diuretic action, mainly through increase in the rate of general blood flow; (5) it causes a constriction of the coronary blood-vessels and diminished flow through the walls of the heart.

As shown by Cushny, the action of digitalis may be divided into three stages, characterized by the following phenomena.

I. **Therapeutic Stage.**—Slowing of entire heart, increase of blood-pressure, increase of systolic output and of cardiac tonicity, peripheral vasoconstriction, dilatation of coronary arteries, slowing of conductivity.

II. **Stage of Irregularity.** The heart-rate becomes somewhat accelerated and irregular. Cushny thinks that this irregularity is due to the fact that, besides following impulses from the auricles, the ventricle begins to beat with a rhythm of its own. A pararrhythmia thus sets in, and the two independent rhythms occasionally produce interference and prolonged pauses. In this stage the blood flow becomes slowed, although the output of individual systoles is much increased.

III. **Stage of Incoördination.**—Extreme irregularity of both auricles and ventricles has now set in, both beating independently of one another

(absolute heart-block). The blood flow has now markedly slowed and beats become irregular in force and rhythm. Death sets in.

Action of Digitalis on the Coronary Arteries.—Another effect of digitalis, namely, marked constriction of and lessened blood flow through the coronary vessels, has been demonstrated by Oswald Loeb upon the excised heart. This is due mainly to the digitoxin, and occurs even at a time when the size and force of the cardiac contractions are definitely increased. Although Dr. G. S. Bond in the writer's laboratory has been unable to obtain any such change in flow through the coronary arteries of the dog's heart in situ after administration of digitalis and strophanthus, there is considerable clinical evidence that digitalis sometimes does distinct harm in patients whose myocardium has undergone extensive fatty degeneration, or cardiosclerosis, or whose arteries are sclerotic. Strophanthin produces the same effects though in less marked degree.

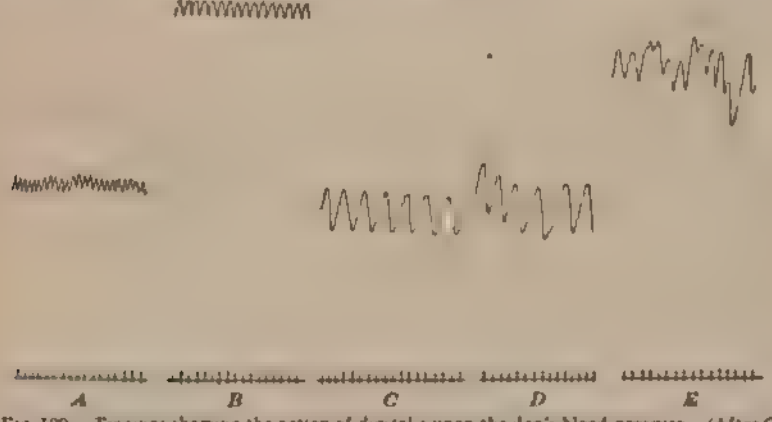


FIG. 129. Tracings showing the action of digitalis upon the dog's blood-pressure. (After Cushny.) *A*, normal. *B*, therapeutic stage with increased blood-pressure and moderate slowing of the pulse, but quickened blood flow. *C*, excessive inhibition causing low blood pressure and slowed circulation. *D*, still further slowing, with slight arrhythmia. *E*, third stage, irregularity with further rise of blood-pressure from excessive vasoconstriction.

Effect on the Blood-pressure.—The rise of blood-pressure due to digitalis is in part due to the increased force and output of the heart, in part to the constriction of the peripheral and, especially, the abdominal blood-vessels. The velocity of blood flow (as shown by product of pulse-pressure × pulse-rate[1]) is usually increased when this effect is brought about (Fellner, Fraenkel). Strophanthus causes less vasoconstriction than digitalis, and hence usually affects the minimal pressure less than the maximal, but increases the velocity of blood flow without causing so great a strain upon the heart. Unfortunately, the preparations of strophanthus are less reliable for continuous action.

Occasionally it is found that both digitalis and strophanthus actually lower the maximal blood-pressure. This occurs especially in the cases where the circulation through the medullary centres is impaired by venous stasis or arteriosclerosis, or failure of the heart,

[1] Janeway has reported cases in which digitalis produced great improvement without increasing P P x P R. Considering the error which may be involved in this calculation, such exceptions are not surprising (see page 24)

and the high blood-pressure is merely the result of general reflex vasoconstriction from the ischæmia of the centre (high-pressure stasis). When the force of the heart is increased and the blood passing through the centre is better aerated, the vasoconstrictor influence is no longer exerted and the general blood-pressure then falls.

Effect of Digitalis on Tonicity.—Clinically the most important action of digitalis is its effect upon the tonus of the cardiac muscle, in preventing and in overcoming dilatation, and it is in dilated hearts that the beneficial action of digitalis is most pronounced. François-Franck (1882) demonstrated that the administration of digitalis did away with the transitory functional tricuspid insufficiency which resulted from stimulation of the vagus. Cushny and Cameron have shown marked increase in tonicity, as shown by diminution in cardiac volume. Moreover, Cloetta has demonstrated that the prolonged administration of digitalis prevents the heart from dilating in experimental aortic insufficiency (positive intraventricular pressure during diastole). The hearts of animals which have been treated with digitalis are smaller and stronger than those which have not been so treated (see page 377). Colbeck, Gossage, and others have also emphasized the importance of this effect on cardiac tonus.

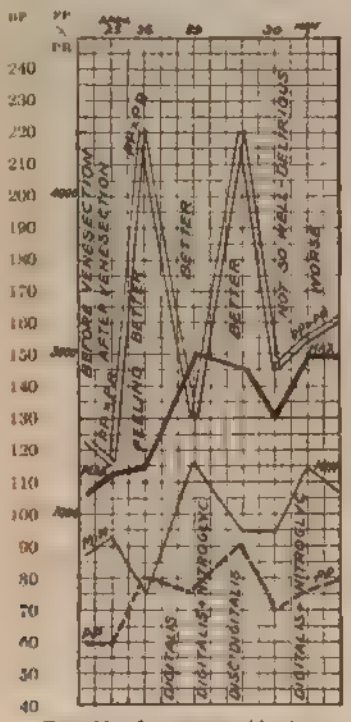


FIG. 130.—Variations in blood-pressure in a patient under the influence of digitalis and nitroglycerin. *MAX* maximal blood-pressure. *MIN* minimal blood-pressure. *PP* pulse-pressure. *PP* × *PR* pulse-pressure × pulse-rate. *DISC. DIGITALIS*, discontinue digitalis.

CHOICE OF DIGITALIS PREPARATIONS.

The first question arising is, What form of digitalis to administer? Numerous attempts to isolate and administer the purified active principle of digitalis have demonstrated that, though several such substances (digitalin, digitoxin, digitalein, digitonin) have been used, digitoxin comes closest to the crude drug. Unfortunately, pure digitoxin is insoluble in water and in the tissue juices, but becomes soluble in the presence of digitonin. The mixture of the two substances known as "digalen" (Cloetta) or "soluble digitoxin" is on the market, and is widely used, especially for intravenous injection, when rapid effects are desired. Digitalin, especially the so-called "German" digitalin, has been much more widely used than digitoxin, but, as is also Kiliani's digitalinum verum, is far less certain and less active.

However, the recent critical studies of Albert Fraenkel have demonstrated that the crude digitalis is at least as satisfactory as any of its derivatives, provided its toxicity (lethal dose for a frog) has been determined and the therapeutic dose standardized accordingly. This is all important, since digitalis leaves from different sources vary greatly in their content of digitoxin, digitalin, etc., and a mere knowledge of the weight of powdered leaves, which is all that is necessary for the pharmacopœial preparations,

gives no idea of the actual potency of the drug. Fraenkel also found little or no difference between digitoxin and crude digitalis as regards the time at which their effects set in, both becoming manifest in twelve to twenty-four hours after administration by mouth or intravenously. The absence of immediate effects from digitalis preparations may be obviated by the use of strophanthin intravenously. This drug acts with great certainty and its action usually sets in within less than half an hour, so that it is very useful in cases where immediate effects are desired. Fraenkel recommends a single dose of strophanthin intravenously to be followed within twenty-four hours by digitalis by mouth, so that the effect of the latter may begin as that of the strophanthin wears off. Of course if the patient has recently received digitalis, strophanthin should not be used for fear of cumulative effect.

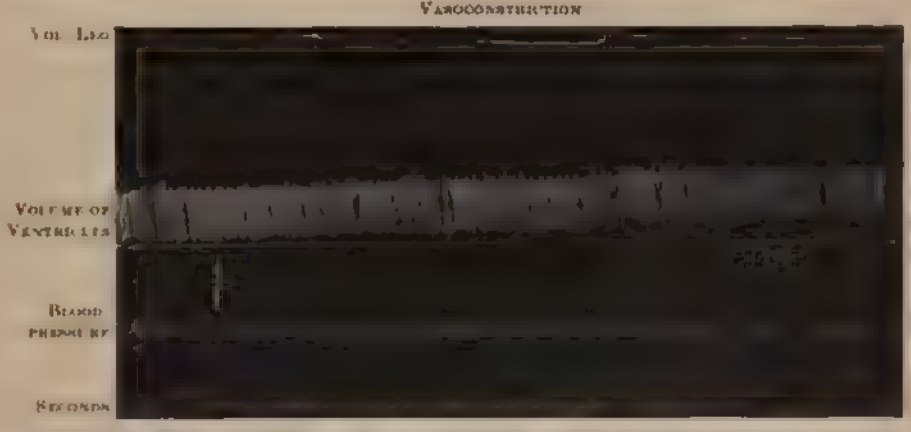


FIG. 131.—Effect of digitalis on cardiac tonicity in the dog. (Experiment by Dr Cameron.) Upper curve, volume of the hind leg, taken with a plethysmograph showing vasoconstriction; second curve, volume of the ventricle; lower curve, blood pressure taken with the Hurthle membrane manometer. Time in seconds. A second dose of 1 c.c. of digitalis injected into the jugular vein at the arrow. The effect upon tonicity, T+, outlasts both the rise in blood pressure and the vasoconstriction.

An example of Fraenkel's remarkable therapeutic effects may be quoted here.

Patient, aged 57, male, admitted to Strassburg Hospital November 17, 1905. Had rheumatic fever in 1869 and again in 1886. Palpitation when at work, and occasional swelling of legs since 1900. Drinks considerably.

Present Condition.—Considerable oedema of legs, thighs, and scrotum. Moderate ascites. Dulness and diminished breath sounds over right base. X-ray shows heart shadow enlarged to left and right; dynamic dilatation of aorta. Pulse irregular, 104 per minute, maximum pressure 180. Pulv. fol. digitalis 0·1 Gm. (1½ gr.) three times a day brought pulse down to 80 in 4 days, to 76 in 6 days, increasing diuresis from 2000 to 4500 and 5800 respectively.

Another attack of pain in joints on December 1, left elbow "improved" on December 11. Returned January 25, 1906. Oedema as before, ascites marked—abdomen 108 cm. in circumference. Liver palpable four fingers' breadth below costal margin. Spleen palpable. Dulness and diminished fremitus over base of right lung. Heart dilated more than before; impulse not palpable; first sound at apex reduplicated; second accentuated, especially over pulmonic area. Slight gallop rhythm. Heart action rapid and irregular. Marked orthopnoea and very distressing cough. Patient has had no sleep for several nights.

The following table shows the effects of the intravenous administration of strophanthin upon his blood-pressure, urine output, and symptoms. The product of pulse-pressure and pulse-rate furnishes a very rough index of the velocity of blood-flow.

Time.	Blood-pressure Max.	Blood-pressure Min	Pulse.	Pulse-pressure ÷ Pulse-rate	Urine in 24 hours	
Jan. 27, 10 30	268	206	92	5640	1000	Dyspnœa increasing.
10 40	286	206	84	7490		Pulse feels larger.
10 45	.	.	1 mg. strophanthin intravenously			
11 00			Diuresis begins			
Jan. 28	260	156	80	7904	*6050*	No feeling of constriction. Sleeps well.
Jan. 31 ...	Œdema almost disappeared.					
Feb 1	Œdema completely disappeared					
	Patient makes uninterrupted recovery with no further medication.					

PRECAUTIONS AND METHODS OF ADMINISTRATION.

Flavoring.—Gastric disturbances, such as nausea and vomiting, occasionally result from the administration of digitalis preparations or derivatives. This is in part due to the direct irritating action upon the gastric mucous membrane and in part to the extremely unpleasant taste and after taste of the drug. In order to obviate the former the drug should always be given in a large quantity (at least half a tumbler) of water. The intensely disagreeable taste of the digitalis and strophanthus preparations may be disguised by the addition of bitter orange peel (tinctura aurantii amari), compound tincture of either gentian or cardamom, or tincture of quassia or calumba. It may also be given in albumin water flavored with lemon so that its taste is barely noticeable. The use of any of these disguises greatly lessens the discomfort of the patient and frequently minimizes the gastric disturbances resulting from the drug.

Rectal Administration.—When the gastric symptoms persist in spite of these precautions, the drug may be administered per rectum, being given in 100 c.c. physiological salt solution with a little starch. This method is very satisfactory (Janeway). Dr. Finley informs the writer that he has seen the pulse-rate slowed and the patient's condition greatly improved within two hours after the administration of digitalis per rectum, whereas the effect rarely follows administration by mouth in less than twenty-four hours.

Period of Administration. In the administration of digitalis it is important to obtain a definite effect and yet not to push the drug beyond the first stage of its activity,—that of slowing and increase in size of the pulse, and to avoid the onset of the second stage, i.e., of irregularity. Since different hearts vary in their susceptibility to digitalis, and since, on the other hand, the drug begins to act only after twenty-four hours and may have a cumulative effect, this task is by no means easy. To avoid the onset of toxic effects various routine methods may be resorted to. Thus, Professor Osler and other authorities recommend giving the drug in "courses" consisting of eight doses of 15 minims of the tincture (0.1 Gm. or 1½ gr digitalis) every four hours. The course is to be repeated if necessary. It may be said that this method often falls short of the effect or brings it about too slowly. The writer has found it very satisfactory to order "0.3 c.c. (5 minims) of

the fluidextract three times a day until the pulse-rate reaches 80, when it should be discontinued without the necessity of a special order." Albert Fraenkel suggests giving several strong doses equivalent to 0.1 Gm. (2 gr.) of powdered digitalis (about twice the usual dose, 2 c.c. or 30 minims of the tinctura digitalis) to insure prompt effect (slowing of the pulse), and thereafter dropping to steady dosage of .03 Gm. (½ gr., 0.5 c.c., or 7½ minims of the tincture) to prevent cumulative but retain the therapeutic effect. This seems to be the most satisfactory method, since it insures not only the immediate but a permament effect. Indeed in many chronic cases "the strength of the heart begins to fail a short time after leaving off the digitalis. Here the continuous use of digitalis (0.05 Gm. or 1 gr. digitalis), as recommended by Kussmaul, Naunyn, and Groedel, for months and even years, has an admirable effect in keeping the cardiac activity at its necessary height" (Romberg).

Digitalis and Nitrites.— In many cases digitalis and nitroglycerin, sodium nitrite, or erythrol tetranitrate may be given together with great advantage (J. O. Hirschfelder). This combination of drugs does more than merely annihilate the constrictor effect of the digitalis, for the nitrites also increase cardiac tonicity and the two drugs unite in bringing about this beneficial effect. Moreover, it is a well-known principle in therapeutics that the combined effect of two equivalent doses of drugs having a common action is often greater than would be produced by using double the dose of either one. As will be seen, this combined action is particularly important in the treatment of aortic insufficiency.

ARRHYTHMIA AND HEART-BLOCK CAUSED BY DIGITALIS.

Mackenzie and later Hewlett have investigated the nature of arrhythmias which have been produced clinically by slight cumulative action of digitalis. They found two forms:

First, the ventricle occasionally fails to respond to contractions of the auricle (partial heart-block). V. Tabora has found in animals that this block is brought about mainly by stimulation of the vagi. If the vagi have been sectioned or paralyzed with atropine, it appears only after a much greater dose has been administered. Hence this digitalis block may be regarded as belonging to the first stage of digitalis effect. To obviate this, Hewlett recommended giving atropine along with the digitalis, a combination suggested by Cushny but discarded by him in favor of digitalis and sparteïn, a drug which paralyzes the vagus without the unpleasant action of atropine (Cushny and Matthews). Neither of these combinations has been used extensively, and, moreover, Cameron's experiments show that atropine prevents digitalis from improving the cardiac tonicity, and hence robs it of its most important effect. The second form of irregularity following digitalis is the occurrence of ventricular extrasystoles, such as were observed in animals by Cushny. As stated by this observer, this effect belongs to the second stage of digitalis action, and accordingly is a more urgent sign for discontinuing the digitalis than is even the partial heart-block.

ADMINISTRATION OF DIGITALIS IN WEAKENED HEARTS.

The relation of digitalis to the arrhythmias has recently been investigated by Dmitrenko, who claims that drugs of this series are always contraindicated in cases where the heart is irregular. This is certainly an extreme view. Hering has shown that certain irregularities, due to extrasystoles arising in the ventricle, disappear under the use of digitalis. Mac-

kenzie has shown that where the irregularity arises in the auricle digitalis may sometimes do positive harm by diminishing conductivity; but this is comparatively rare. On the other hand, da Costa, Leyden, and the later writers have shown that in the permanent irregularities digitalis does not cause the arrhythmia to disappear, but usually increases the force of the individual contractions, causes them to become less unequal, and increases the velocity of blood flow. The effect depends largely upon the condition of the heart and its susceptibility to the drug. In general, the more diseased the organ the more sensitive it is to the action of small quantities. A very weak heart with intense myocardial change may therefore pass to the second stage of digitalis action under smaller doses than would bring about a physiological effect in one whose fibres were less intensely degenerated. The effect of digitalis upon the patient must always be carefully watched, and if the rhythm becomes more irregular it must be discontinued. On the other hand, where the myocardium is reduced to small amount in extreme fatty or fibrous myocarditis, the increased strain (and perhaps also the coronary vasoconstriction) caused by digitalis is often too great, and the failure of the heart is increased and the drug does distinct harm. At present no absolute rule can be laid down for the border-line cases in which there is doubt, except that when an irregularity is present, especially one which has its origin in the auricle or great veins, digitalis should be used only to treat cardiac dilatation, and even then with great hesitancy and extreme precaution. When any disturbance of conductivity occurs, digitalis is absolutely contraindicated.

HALLUCINATIONS FROM DIGITALIS.

Another toxic effect of digitalis lies in the production of mental symptoms, delirium and delusions, through its action on the central nervous system (Duroziez, Hall, see page 160). The onset of these symptoms therefore constitutes a contraindication to continuing the drug.

CHOICE OF DRUGS.

As between digitalis and strophanthus, the choice lies with the former except in the following conditions: (1) when rapid action is needed, in which case strophanthin should be given intravenously; (2) in cases of myocardial weakness or fatty degeneration, when it is important not to increase peripheral resistance, (3) in cases of aortic insufficiency and of mitral stenosis, where the same is true; (4) in some cases with aneurism and broken compensation.

Besides digitalis and strophanthus numerous other drugs and their derivatives enumerated above have been introduced, but none seems to have any decided advantages which warrant supplanting these two.

SUMMARY OF CLINICAL APPLICATION OF DIGITALIS.

Professor Osler's epigram, "Broken compensation is the signal for digitalis," about summarizes the use of the drug. Its applicability in individual diseases will be discussed under the separate chapters, but in general

it may be said to be useful in three classes of conditions: (1) in cases where compensation is broken; (2) in cases where acute dilatation is present and has persisted after rest and other modes of treatment; (3) in cases with persistent or distressing tachycardia, which does not yield to other means. It is in general absolutely contraindicated. (1) in cases with heart-block; (2) where the amount of heart muscle has been diminished by fibrous or fatty myocarditic changes, or in a case in which digitalis has been known to fail already. It should be used with caution: (1) in arrhythmias due to disturbances arising in the auricles or sinus region; strophanthus is equally contraindicated; (2) in cases with coronary sclerosis, owing to the constricting action upon those vessels, strophanthin is here less undesirable.

STRYCHNINE

PREPARATIONS.

Strychnine (strychnina) is an alkaloid obtained from nux vomica. Tinctura nucis vomicæ contains 2 per cent. extract of nux vomica and is assayed to contain 0.1 per cent. strychnine. It is useful more as a stomachic bitters than as a cardiac stimulant. Dose, 1–2 c.c. 15 to 30 minims.

Strychninæ sulphas contains 5 molecules of water of crystallization and 78 per cent. of strychnine. Soluble in 3 parts of water. Average dose, 0.0015 Gm. ($\frac{1}{40}$ gr.)

Strychninæ nitras is soluble in 42 parts of water and 120 parts alcohol. Dose, same as sulphate.

PHARMACOLOGICAL ACTION OF STRYCHNINE.

There are many cases in which the circulation is beginning to show some signs of slight weakening and yet where it does not seem necessary to use digitalis. In these cases other drugs are resorted to, in America usually strychnine,[1] in Germany usually camphor; both apparently yielding good clinical results. It must be added, however, that according to most pharmacologists strychnine has no effect whatever upon the heart and produces the rise in blood-pressure only by the vasoconstrictor action.

Effect on Cardiac Tonicity.—Dr. P. D. Cameron has recently investigated the subject under the writer's direction, and has found in the dog that strychnine in doses of .00003 Gm. per kg. or $\frac{1}{15000}$ gr. per lb., corresponding to .002 Gm. ($\frac{1}{35}$ gr.) hypodermically for a man, always produces an increase in tonicity of the heart muscle, though without affecting the force of the beat or markedly changing maximal pressure. Mean and minimal pressures are usually slightly increased (by 10–15 mm. Hg) and pulse-rate a little slowed. Larger doses increase the systolic output, raise the blood-pressure, slow the heart, and increase the tonicity.

Clinical Effects. In view of the wide-spread and often indiscriminate use of this drug, it is important to realize exactly its clinical use before prescribing it. As has been stated, strychnine stimulates both vasoconstrictor and vagus centres, hence raises the blood-pressure and slows the pulse-rate. These effects, however, have been observed mainly in animals, and few exact clinical studies have been made upon man in connection

[1] In view of the wide use of strychnine in heart diseases in English-speaking countries, it is quite striking that this drug is not mentioned in connection with therapy of the circulatory system in such extensive German text books as those of Romberg and Heinz.

with observations of the change of blood-pressure. Briggs and Cook, who were most enthusiastic over the use of the drug, did not obtain rises of blood-pressure exceeding 10 mm. Hg from doses of 1 to 6 mg. ($\frac{1}{60}$ to $\frac{1}{10}$ gr.) and in no case slowing of the pulse. Cabot and F. P. Drayer, on the other hand, failed to note any changes whatever in many cases. The writer has made a considerable number of observations, determining the blood-pressure with the Erlanger apparatus. He injected strychnine in doses which

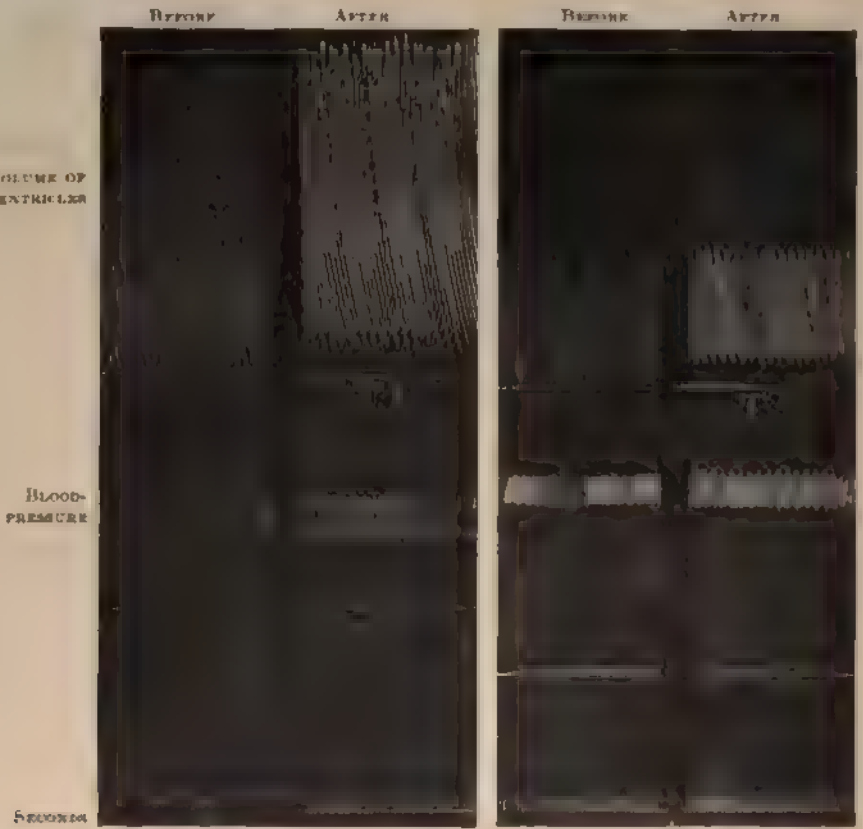


Fig. 132. Curve showing the effect of strychnine upon cardiac tonicity. Experiment by Dr. Cameron. Lettering as in Fig. 131. Fig. I shows increase in systolic output and maximal blood-pressure. Fig. II shows increase in tonicity with a diminution in the systolic output and a fall in the minimal but no change in the maximal blood pressure. The effect upon tonicity is the most constant effect of the drug.

rose to 15 mg. ($\frac{1}{4}$ gr.) hypo. without obtaining any effect upon maximal or minimal pressure, pulse-rate, or rate of respiration, and from single doses scarcely any increase in reflexes. These tests were made upon hearts which were not dilated, and hence no effects upon tonicity could be noted.

Since the rank and file of English and American physicians entertain an almost superstitious belief in the efficacy of this drug, it is evident that effect in each case should be controlled by blood-pressure determinations. Cameron's experiments upon animals have shown that a distinctly bene-

ficial effect upon tonicity may be obtained with but little change (5-10 mm.) in the maximal blood-pressure, but that often when these changes are very slight the effect may be much more distinctly shown by a rise in the mean or minimal pressure. Both should be carefully watched in cases in which strychnine is given, and the dose should be sufficient to be effective. If no effect is obtained it should be discarded for some more potent drug.

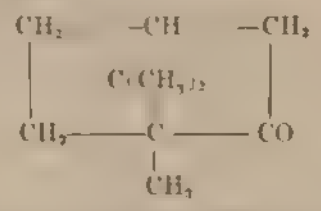
INDICATIONS FOR STRYCHNINE.

It is probable that strychnine is of particular value in the disturbances of respiration following extreme heart failure, such as Cheyne-Stokes breathing, cardiac asthma, etc., as claimed by Eyster; and in such cases it should be given whether digitalis is being administered or not.

By virtue of its stimulating action upon the vasomotor centre, strychnine is particularly indicated in cases in which this centre is beginning to fail. This is particularly the case in all infectious diseases, in many cases of neurasthenia, in mild shock, in some cases of anæmia, asthenia, and in many convalescents. Strychnine should be used not to replace digitalis, but may be given as a prophylactic to prevent the heart muscle from wearing itself out upon a relaxed vascular system. When the heart muscle once shows signs of giving way, when marked cardiac dilatation, etc., have set in, its period of usefulness is over. Small doses of digitalis will then do the same work better and will do more. It is also valuable when given along with digitalis. The value of strychnine again becomes manifest in the later stages of heart failure through its action as a stimulant for the respiratory centre. Eyster believes that it is particularly useful in warding off Cheyne-Stokes respiration and also in the treatment of the latter. It is probably still more useful in cardiac asthma, more as a prophylactic measure in maintaining the activity of the respiratory centre than in stopping individual attacks; and it may also prove of value in warding off the distressing dreams that result from mild asphyxia during sleep, as well as the attacks of tachycardia and other unpleasant conditions which may occur as the result of waking "with a start" (asphyxia during sleep).

CAMPHOR

Camphor is a white substance, soluble in alcohol, ether, and chloroform, whose structural formula is—



Average dose 0.12 Gm. (2 gr.), best given as linimentum camphoræ (camphorated oil), which contains 20 per cent. of camphor dissolved in cottonseed oil. Average dose 0.75 to 2.0 c.c. (20 to 45 minims), available for hypodermic use or by mouth.

Spiritus camphoræ, a 10 per cent. solution of camphor in alcohol. Dose 1 c.c. (15 minims).

Camphor, like strychnine, is a stimulant to the vasomotor centre, but, according to Cameron, does not seem to have so pronounced an effect on tonicity. Like strychnine it also varies in its effects on different individuals. Some persons require doses twenty times as large as do others before an effect sets in, especially when the drug is given by mouth. Camphor is most important for its use in shock. It is given deeply into the muscles in order to avoid subsequent inflammations. It is not so valuable for continuous use. As recently shown by Winterberg, Schigmann, and Gottlieb and Magnus, camphor has also a very distinct action upon the heart muscle, causing the fibrillating excised heart to revive from fibrillary contractions. After camphor has been administered to a dog the ventricle (*in situ*) can be thrown into fibrillary contractions by weak faradic stimuli and yet recover. On the other hand, a direct therapeutic effect upon the heart muscle in man has not yet been proved.

CALCIUM SALTS.

Calcium chloride and other salts of calcium have been recommended recently as cardiac stimulants by Lauder Brunton and other English clinicians. Although the action of calcium upon the excised heart is indisputable, its effect upon the heart *in situ* has been supposed to be too transitory to be of practical value. The writer has been unable to find any effect upon the maximal and minimal blood-pressures and pulse-rates of a number of cases of typhoid fever who were receiving calcium lactate in sufficiently large doses to hasten coagulation. Sladen also found in a large variety of cases that calcium lactate had no effect upon pulse-rate or blood-pressure. On the other hand, in animals calcium chloride has an effect, especially upon tonicity, which closely simulates that of strychnine. Injection of considerable quantities directly into the cavities of the heart revives that organ as nothing else appears to do. The writer has found that in some cases dogs' hearts that had actually stopped beating and even lost their mechanical irritability revived to such an extent as to resume a regular rhythm with a moderately high blood-pressure. The matter is, however, still in the experimental stage.

CAFFEINE.

From the results of experiments upon animals, caffeine would take rank next to digitalis in cardiac therapy. Like digitalis it acts upon the cardiac muscle, increasing the size and force of the contraction; like digitalis it has a vasoconstrictor action, and raises the blood-pressure by bringing about constriction of the peripheral blood-vessels. It is therefore particularly valuable in conditions of collapse and shock. In this regard it is more reliable than camphor (Romberg) or strychnine.

On the other hand, caffeine does not exert a constricting action upon the coronary arteries (O. Loeb), and hence is not contraindicated in cases of coronary sclerosis. Upon the pulse-rate caffeine exerts a variable effect, in relatively small doses (0.1 Gm., 2 gr.) slowing the pulse by stimulating the vagi, in larger doses accelerating. The acceleration is apparently due to direct action upon the heart muscle, since it occurs also in the excised heart when caffeine is added to the Locke's solution. However as regards the effects of a given dose, there is the greatest variation among different individuals, some persons being extremely sensitive to small doses, others extremely resistant. Even in the same individual tolerance varies. Thus a considerable degree of tolerance may be developed by the constant use of coffee, so that three or four cups (0.15 to 0.2 Gm., 3 to 5 gr. caffeine) a day may be taken with no symptoms whatever. Thus in a case under the writer's observation, after several months of absolute abstinence from coffee, marked palpitation, tachycardia, and sleeplessness resulted from a single cup in twenty-four hours; a few weeks later one cup and after a few months two cups could be taken without any apparent effect.

Unfortunately, the therapeutic use of caffeine is often accompanied by palpitation, sleeplessness, and even nausea, vomiting, vertigo, and delirium, which occur with particular ease in cases with cardiac disease. In using

caffeine one is therefore usually in a dilemma between a hypersensitiveness and an habituation. Unfortunately, the palpitation and discomfort usually set in at about the same point as the therapeutic effect, or even earlier; but there are certainly many cases in which this is not the case, and in which caffeine is a valuable therapeutic agent.

THEOBROMINE.

Theobromine has a much less effect upon the cerebral cortex and upon the vasomotor centre than caffeine, but has a very strong diuretic action. As shown by O. Loeb it possesses a much more powerful action in dilating the coronary arteries of the excised heart. Upon the heart *in situ* its action does not seem to be pronounced. Indeed, G. S. Bond, in the writer's laboratory, has been unable to detect any effect upon the outflow from the coronary veins as the result of intravenous injection of agurin (theobromine sodium acetate). The stimulating action of theobromine upon the heart muscle, though not as intense as that of caffeine, is still very marked. It has therefore been recommended as a cardiac stimulant, particularly by the French clinicians, who found it of considerable value in the weak hearts of fatty individuals. Kaufmann and Pauli, Brewer and v. Leyden recommended the use of theobromine in attacks of angina pectoris (stenocardia). Pineles advises theophyllin. Pal has found that theobromine is occasionally useful in the treatment of vasomotor crises, but that it often fails in cases where iodine and potassium thiocyanate help. Romberg is not able to detect any beneficial action of theobromine apart from its diuretic action. In using theobromine it is preferable to use those compounds which are free from salicylates, since this radical has a certain depressant action upon the heart and an irritant action on the kidneys. Acettheobromine sodium ("agurin") and acettheocin sodium are therefore preferable to theobromine sodium salicylate ("diuretin").

ACONITE.

PREPARATIONS.

Aconitum, the dried tuberous root of aconitum napellus, collected in autumn, and yielding not less than 0.5 per cent. aconitin. Dose 0.05 Gm. (1 gr.).

Tinctura aconiti, U.S.P., now represents 10 per cent. of the crude drug, formerly stronger. It is the most certain and most stable of all the aconite preparations. Dose 0.6 c.c. (10 minims).

Aconitina, the crystalline alkaloid. Dose 0.00015 Gm. (0.15 mg. or $\frac{1}{400}$ gr.). It is so irritating that it is usually preferable to prescribe the simple tincture of aconite, since this is assayed according to the last pharmacopœia.

Pharmacological Action.—Aconite has three pharmacological actions upon the circulatory system: (1) it stimulates the vagus promptly and to a high degree; (2) it diminishes the size and force of the cardiac contraction, and also accelerates the heart when this organ is liberated from the action of the vagus centre; (3) it slightly stimulates the vasomotor centre in very small doses. However, it also diminishes the activity of the respiratory centre, and may thus bring on dyspnœa.

Therapeutic Uses.—When carefully given in therapeutic doses aconite slows the heart by stimulation of the vagus, and has little action upon the

heart muscle. It is therefore of value in the acceleration of the pulse in fevers, where the heart muscle itself needs no stimulation and the heart needs slowing. Owing to the variability of the tincture under the old pharmacopœia, the use of aconite has fallen into disrepute, and enough time has not elapsed since the adoption of the last pharmacopœia (1900, adopted in 1905) for its real utility in physiological therapeutics to have been investigated. There is no doubt that it is of value in many cases of tachycardia, especially those of nervous or postfebrile origin. Da Costa, in 1864, found it of some value for the tachycardia of acutely overstrained hearts, but particularly useful when given with digitalis. This combination contains two drugs; both stimulate the vagi, the one tends to diminish, the other to increase the force of cardiac contraction. If the latter effects balance each other it may be possible to obtain in this way the purest and most intense action in slowing of the pulse.

It is certain that the simultaneous use of two drugs having certain actions in common often brings about an effect not obtainable with either drug alone, but since the reaction against the polypharmacy that reigned during the middle of the last century the tendency has been toward the use of single drugs. There is no doubt that much can be learned in the treatment of cardiac diseases by judicious combinations along the lines mapped out by pharmacological experiments, just as is now found with hypnotics, analgesics, and purgatives. This is radically different from the ancient polypharmacy, in which heterogeneous drugs were mixed without regard to their action or antagonism.

ADRENALIN

Adrenalin (suprarenin, epinephrin), the active principle of the suprarenal gland, is also used occasionally to raise blood-pressure by its constricting action upon the peripheral blood-vessels and slight stimulating action upon the heart, but its action lasts only from one to two minutes and hence it is of little value, except to tide over a sudden failure until some other drug can become active.

ERGOT.

Ergot has been recommended by some writers for its vasoconstrictor action exerted through stimulation of the vasomotor centre. It also stimulates the vagal centre. Cronyn and Henderson have found that these effects are very uncertain when the drug is given by mouth, but occur quite uniformly when it is given intravenously. Since this is rarely necessary, the use of ergot may be confined to patients with vasomotor failure, in which, like adrenalin, it is used as a last resort.

NITRITES AND NITROGLYCERIN

PREPARATIONS. (PHARMACOPŒIAL, U. S. P.)

Amyl nitrite (amylis nitris), a liquid containing about 80 per cent. of amyl nitrite. Average dose 0.2 c.c., 3 minims inhaled). Usually to be had in pearls, each pearl containing one dose.

Nitroglycerin CH_2ONO_2
$|$
$CHONO_2$
$|$
CH_2ONO_2

is sold in tablets of varying size, usually one tablet containing $\frac{1}{100}$ gr. (0.6 mg.). However, in tablet form the nitroglycerin is liable to undergo more or less rapid deterioration, and hence administration in this form is unreliable. It is best given as spiritus glyceryhs nitratis (spiritus glonoini), a 1 per cent solution of nitroglycerin in alcohol, which should be freshly prepared from a 10 per cent stock solution. Initial dose 0.05 c.c. (1 minim), increasing if necessary 1 minim at a time.

Sodii nitris (sodium nitrite), $NaNO_2$, a white fused mass, very deliquescent and slowly becoming oxidized to sodium nitrate on exposure to the air, thus becoming useless. Dose 0.06–0.12 Gm. (gr. 1–2).

There are also several non-pharmacopœial nitrates which are very satisfactory. Erythrol tetranitrate, CH_2ONO_2-$CHONO_2$-$CHONO_2$-CH_2ONO_2, has about the same action as nitroglycerin, except that it acts more slowly (action lasting three to four hours). Sold as tablets, each containing .03 Gm. (½ gr.). Dose one or two tablets every four to six hours. In the cases in which the writer has used it erythrol tetranitrate has been very efficient and satisfactory.

ACTION OF THE NITRITES.

In practical therapy the nitrites are drugs of great importance. In animals they are found to act upon the muscles and nerves of the blood-vessels to bring about an intense vasodilatation, thereby diminishing the resistance to blood flow and lessening the resistance to the action of the heart. As far as can be judged from the studies of O. Loeb, they do not influence the vasoconstrictors of the coronary arteries unless present in concentration which is absolutely toxic to heart muscle. G. S. Bond has found that the outflow through the coronary veins of normal dogs is decreased rather than increased by nitroglycerin and amyl nitrite. It is therefore questionable whether these drugs ever bring about dilatation of the coronary arteries, as has been supposed from their efficacy in angina pectoris.

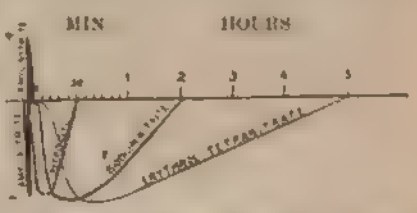


Fig. 133.—Effects of drugs of the nitrite series upon the blood-pressure of man. Scheme representing the findings of Hewlett and Matthews. M/N = minutes.

The relation of the various nitrites to one another as regards rapidity of action is shown in Fig. 133. The effect of amyl nitrite sets in within a minute and passes off within five minutes, that of nitroglycerin lasts from about the seventh to the twentieth minute after administration, sodium nitrite from the fifteenth to the thirty-fifth, while erythrol tetranitrate begins to exert an effect only after about fifteen to thirty minutes, but this continues for three to four hours.

Amyl Nitrite. Hewlett has recently made a careful clinical study of the effects of amyl nitrite inhalation and found, (1) an immediate fall of maximal pressure, average 13 mm. Hg, lasting less than forty seconds and accompanied by a less fall of minimal pressure and an increase of pulse-rate. This is followed by a secondary rise (about 28 mm.) of maximal pressure to considerably above the original height, accompanied by a less marked rise of the minimal pressure and by a return of pulse-rate to the normal. These changes in blood-pressure correspond to an increased systolic output and increased force of heart beat (augmentor effect), and Hewlett was able to see with the fluoroscope that, as the action of the heart slowed down the excur-

sions of the left ventricle became wider by one-half centimetre, but they soon returned to normal." In other words, besides being a vasodilator amyl nitrite is a very active cardiac stimulant, more rapid than any except adrenalin. These findings accord well with the results of Cameron on dogs, that nitroglycerin both increases cardiac output and cardiac tonicity to a marked degree. Relaxation of the peripheral blood-vessels under the influence of the amyl nitrite, as shown by the plethysmograph, was present throughout all Hewlett's experiments in spite of the peculiar variations of blood-pressure. The vasodilatation reaches its maximum within the first minute and very gradually subsides after the second, but a definite effect is still noticeable ten or twelve minutes after. In older persons Hewlett found that the pulse rate often did not change, probably owing to the absence of the tonic activity of the vagus.

The following represent typical effects in normal men as obtained in the very careful clinical investigations of Hewlett and Matthew.

Dose.	Time when action begins.	Average fall in B P	Maximal fall occurs in	Duration.
Amyl nitrite (Hewlett)	15 sec	20	1 min	10 min
Nitroglycerin 05 1 c.c (gr. ¾ ij)	1 min	28	4½ min.	½ hr.
Sodium or potassium nitrate 15 Gm (gr ij)	5 min.	32	11 min	
Erythrol tetranitrate 03 06 Gm (gr ½ i)	5½ min	35	22 min	
Mannitol tetranitrate 06 Gm (gr i)	12	35	100 min	

Nitroglycerin. As to nitroglycerin, there is tremendous variation in its effects upon different individuals. A. Loeb having reported a case of collapse after 0.6 mg. (1/100 gr.), whereas J. Stewart has given 20 grains a day to a single patient.

In some cases it is impossible to obtain a fall of pressure with any ordinary doses. The writer's experience agrees with that of Matthew, that the effects are often lacking in cases of nephritis in which high blood-pressure has persisted for some time. To this might also be added a certain group of arteriosclerotics in which the renal symptoms do not predominate, although it is possible that arteriosclerotic changes may be present in the kidney.

In Prof. J. O. Hirschfelder's wards it was customary to begin with a dose of 1 gtt. (1/90 c.c., ¼ minim) every half hour, increasing 1 gtt. at every third dose until palpitation, headache, or buzzing in the ears warned that the physiological limit had been reached. The next dose was then omitted and a permanent dosage of 1 gtt. less than the dose last given was then kept up. In some cases as much as 1 c.c. (15 minims) of the 1 per cent. solution was given every half hour with only the mildest subjective symptoms, the average permanent dose being 0.3 to 0.6 c.c. (5 to 10 minims). The effect of these doses is very variable.

Effect on the Circulation. A fall in minimal blood-pressure is the most constant, usually accompanied by a rise in pulse-pressure, and the maximal pressure sometimes rising, sometimes falling. Hewlett thinks that there is combined dilatation of the blood-vessels and increased systolic output of the heart. In a series of observations upon the fluctuations of blood-pressure after the administration of these drugs, made with the Erlanger apparatus independently of and some years before those of Hewlett, the writer had noticed effects quite similar to those above mentioned. There seems no doubt, therefore, that, as stated by Hewlett, the beneficial effects of the nitrites in man are due to something more than a simple vasodilatation, and indeed it is possible that the latter may play often even a minor

rôle. Certain it is that in many cases they are ideal drugs to relieve the work of the heart over short periods when the blood-pressure is not already too low to admit of their use. However, it must be borne in mind that individual susceptibilities vary, and the patient should be tested with amyl nitrite, whose effects can be controlled, before any other nitrite should be given. When used over long periods of time, moreover, the production of methæmoglobin in the blood may be brought on (shown by the spectroscope, or by a chocolate tint in the blood), which is distinctly harmful and a sign for immediately stopping the use of the drug.

POTASSIUM IODIDE.

Potassium iodide is the drug which is most widely used in the treatment of all forms of arteriosclerosis, and the clinical results are so definite as to render its usefulness certain. The manner in which it exerts this beneficial action is, however, much less definitely known. It was at first supposed by Potain and others to lower the blood-pressure by some direct action upon the vasomotor or cardiac mechanisms, but this action is slight if any, and the writer does not recall ever having seen high blood-pressure depressed by potassium iodide without the intervention of some other factor. It was then supposed to have some effect in diminishing the viscosity of the blood, as was claimed by Otfried Müller and Inada in Romberg's clinic. A careful persual of their statistics shows that the results were absolutely negative in about half of their cases and within the limits of observational error in the others. Determann, who repeated their experiments, found the effect upon viscosity entirely negative.

It was then claimed by Koranyi and others that potassium iodide prevented the production of adrenalin arteronecrosis, but this claim also fell to the ground when tested upon a very large series of animals by Leo Loeb and Githens.

The pharmacological action of potassium iodide is therefore still to be classed among those mysterious actions termed "alterative."

Some light is thrown upon the action of potassium iodide by the recent studies of Collins and Sachs and Longcope upon the vascular changes due to syphilis. These observers obtained a positive Wassermann reaction in many cases of aortic insufficiency in which there was no other sign of active luetic lesion. In these cases and also in simple arteriosclerosis of luetic origin the potassium iodide probably facilitates the removal of the luetic exudations and thus diminishes the ill effects of the arterial lesion. This would explain why no action can be detected upon the healthy vessel or upon the mechanical factors in the circulation.

It must be admitted that the mode of action is entirely unknown; but, on the other hand, administration of potassium iodide does lessen the symptoms of stenocardia and other painful and disagreeable symptoms in many cases of arteriosclerosis, and may even cause them to disappear permanently. It may therefore be administered with advantage in all cases in which the above-mentioned symptoms arise or even where they are threatened.

Potassium iodide is best given after meals in large amounts (half glassful or glassful) of water or milk. The unpleasant taste may be disguised by a little sherry, elixir of calisaya, or gentian. Dose potass. iodi 0.3 to 2.0 Gm. (gr. v to xxx) (reached by increasing doses).

When not well borne by the stomach or when the heart is very weak, sodium iodide, the iodized fatty acid "iodipin," or new iodized organic acid "sajodin" may be substituted. Their action does not seem to differ much from that of potassium iodide.

POTASSIUM THIOCYANATE.

Another drug which tends to lower the blood-pressure greatly is potassium thiocyanate (KCNS). The use of this drug as a sedative to the nervous system was first suggested by W. Pauli (1903), who believed that he obtained some excellent results in eleven arteriosclerotics and in two cases of heart failure. It was used more carefully by J. Pal (1905), who writes: "I have been able to obtain a good effect from thiocyanate preparations in some cases in which even potassium iodide was without effect. This cyanate often gradually reduces a high blood-pressure, but often brings on *symptoms of intoxication* in arteriosclerotics, especially in those with renal complications. These toxic symptoms are *erythemata* and *mental confusion*, which disappear, as I have found, when the thiocyanate is left off and opium given . . . Diuretin and iodide or thiocyanate are of value (in vasomotor crises) only when administered over long periods."

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VI.

GYMNASTICS AND HYDROTHERAPY.

GYMNASTICS.

FUNDAMENTAL PRINCIPLES.

During recent years gymnastic exercises have come to play a major rôle in the treatment of cardiac diseases. Although this treatment was introduced empirically, its physiological basis is found in the fact, shown by Frank and Hirschfelder, that a strain upon the ventricles which does not exhaust them tends to act as a stimulus which gives rise to more forcible contractions, increases their tonicity, and causes the residual blood (and hence the dilatation) to decrease. The guiding principle is further given by the experimental evidence produced by these writers, that when the strain was excessive it had the opposite effect, and caused weakening of the contractions, diminished tonicity, and dilatation of the heart. (See Fig. 119, page 136.)

In dealing with normal individuals it is observed that the strengthening of every normal individual, the training of every athlete or laborer consists in the habituation of the body, and particularly of the heart, to gradually increasing muscular effort and exercises. (See page 198.) To a great extent, as has been seen, page 129, this consists in securing a greater increase in output of blood at each beat without calling upon any of the accessory nervous mechanism to bring this about. Such exercises have also been used with great success in the treatment of patients with heart failure. It stands to reason that they should not be used at once when the patient is brought in with an acute heart failure; but after a sufficiently long period of rest, when the acute condition has passed off and he can sit up in bed without discomfort, a few of the mildest arm movements may be begun with great advantage. It is often better to train the patient by a few mild passive or resisted movements while he is still in bed than to subject him at once to the strain of getting up for an hour or so after his sojourn in bed. Moreover, many other muscles may be kept in tone, the blood-vessels in the muscles may be kept dilated, and the resistance to blood flow may thus be diminished.

SYSTEMS OF EXERCISE.

In accordance with these facts several systems of exercises have been developed for assisting in the training of the heart. In all of them the crucial point lies in the avoidance of the slightest fatigue, holding of the breath, or increased breathing. Hence the actual result obtained depends more upon the vigilance and intelligence of the physician, nurse, or attendant who supervises the exercises than upon the exercises themselves.

In general the exercises may be divided into four classes:

(1) Passive movements.
(2) Contraction of antagonistic muscles.
(3) Resisted movements.
(4) Mechanical gymnastics.

Passive Movements. These are the mildest possible forms of exercise. The attendant grasps the patient by the hands or feet and moves these members gently and slowly about, while the patient makes no effort at contraction whatever. Such movements have the effect of increasing the circulation of lymph, the absorption of œdema, and, to a certain extent also, of increasing the rapidity of blood flow. It is important to avoid all exercises in which the arms are raised high above the head, since this hydrostatically increases the pressure in the vena cava and may cause momentary dilatation of the heart.

The following exercises or modifications of them may be carried out while the patient is still in bed, provided the greatest precaution is used in their execution.

(1) Arms horizontal, to the front and back to the line of the shoulders.
(2) Arms horizontal in line of shoulders, thence down to the sides of the body.
(3) Arms horizontal, describe circles with hands.
(4) Arms vertically dependent at sides, flex and extend elbows.
(5) Arms dependent at sides, pronate and supinate alternately.
(6) Clinch and open fists.
(7) Legs straight, abduct, then adduct thighs.
(8) Flex and extend knee at side of couch, never raising knee above level of body.
(9) Flex and extend foot at ankle-joint.
(10) Rotate thighs internally and externally.
(11) Execute small circles with feet without raising them more than one foot; legs straight.

Contraction of Antagonistic Muscles.—Substantially the same exercises may be carried out by allowing the patient himself slowly and simultaneously to contract both the muscles concerned in the movement and those which antagonize them,—*i.e.*, biceps and triceps, flexors and extensors of wrist, etc. In this way little movement is made, the pulse-rate is slowed rather than accelerated, and yet a good deal of energy may be expended. The blood-pressure is raised, however. If the patient can be trained to avoid all difficulty in breathing and all discomfort, a good deal of improvement in muscular strength and in cardiac tonicity may be obtained by this method. Its main drawback lies in the fact that the intensity of the exercise is controlled not by the attendant but by the patient, and that the latter is most likely to do more than is beneficial.

RESISTED MOVEMENTS. (SCHOTT MOVEMENTS.)

Probably the most widely used of all the cardiac gymnastics are the passive movements introduced by August Schott of Nauheim. These are generally used in connection with the Nauheim baths. This combination is particularly advantageous and permits at once of all the advantages of mild exercise, of baths, of rest and stimulation to sleep, of psychic sedative, and of the psychic suggestion to the patient that a great deal is being done and a great effort is being made for his welfare.

The Schott movements consist of practically the exercises described above carried out by the patient himself, but with an attendant who makes a slight resistance to each movement. The resistance should be just enough to prevent the movement from being made rapidly, and at no time should it cause the patient any apparent effort or increase his respirations. Each day the resistance may be increased slightly, so that in a short time the patient may be doing a good deal of work without realizing it. In executing the resistance the attendant's mind is kept fixed upon the condition of the patient, and he is consequently more likely to notice over-exertion in the latter than if he were merely supposed to watch him without doing anything himself. In carrying out the Schott movements the following rules are prescribed.[1]

Precautions for Schott Exercises.—(1) Each movement is to be performed slowly and at uniform rate.

(2) No movement is to be repeated twice in succession in the same limb or group of muscles.

(3) Each single or combined movement is to be followed by an interval of rest.

(4) The movements are not to be allowed to accelerate the patient's breathing, and the operator must watch the face for the slightest indications of (*a*) dilatation of the nostrils, (*b*) drawing of the corners of the mouth, (*c*) duskiness or pallor of the cheeks or lips, (*d*) yawning, (*e*) sweating, (*f*) palpitation.

(5) The appearance of any one of the above signs of distress should be the signal for immediately interrupting the movement in process of execution, and for either supporting the limb which is being moved or allowing it to subside into a state of rest.

(6) The patient must be directed to breathe regularly and uninterruptedly, and, should he find any difficulty in doing so, or for any reason show a tendency to hold his breath, he must be instructed to continue, counting in a whisper throughout the progress of each movement.

(7) No limb or portion of the body of the patient is to be so constricted as to compress the vessels and check the flow of blood.

Schott Exercises.—The following is a list of Schott exercises in the order in which they are given. The resistance is moderate and steady, the operator's hand always being applied upon the surface of the extremity toward which the movement is made, even if that entails gliding around it gently during the movement. Usually the operator's hand is at one side of the patient's limb at one phase of the exercise and at the opposite when the movement is reversed.

1. Arms extended in front, palms facing each other. The operator's palms rest upon the backs of the patient's hands. Patient's arms carried backward to line of shoulders, the movement being gently resisted by operator (Fig. 144). The operator's palms are then rested against those of the patient, and the return of the arms in front of the chest is resisted.

2. One arm at side, elbow-joint flexed upward to shoulder, then extended to original position.

3. Arms at side, raised outward till thumbs meet over the head, then brought back to the original position.

4. Hands at level of pelvis in midline, fingers slightly flexed. Arms raised to the vertex of the head, then back.

5. Arms at sides, then raised forward in parallel planes until they are vertical, then moved back. The hand of the operator must glide around the wrist so that it is always applied to antagonize the movement.

[1] Quoted from W. Bealy Thorne.

6. Trunk flexed on hips, knees straight; trunk then extended.

7. Trunk rotated without movement of the feet. Operator exerts resistance against the shoulders.

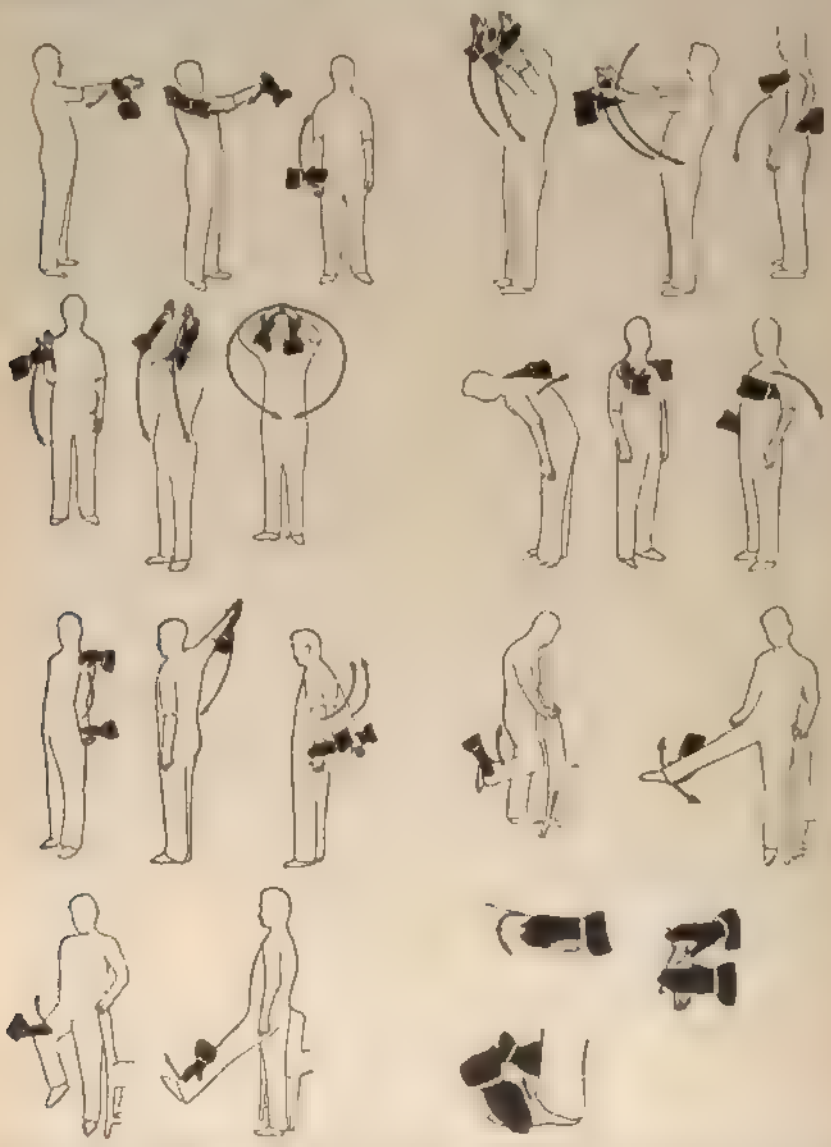

FIG. 134.—Schott resisted movements. (Modified from W. Bealy Thorne.) The attendant's hands are indicated in black, the direction of the movement made by the patient is indicated by the black arrows.

8. Trunk flexed laterally, first to one side then to the other, the movement being antagonized by resistance applied in the axilla, the operator's other hand resting on the hip.

9. Movement like No. 2; fists clinched.

10. Same, but palmar surface of fist turned outward.

11. Arm extended from side, palm down, raised forwards and upwards describing a semicircle until it is raised vertically along side of the ear. The movement is then reversed.

12. Arms at sides, palms inward, moved upwards and backwards in parallel planes.
13. Patient rests one hand on chair or table, raises knee to horizontal, flexing at hip and knee.
14. With one hand resting on table, patient swings extended leg forward and backward from the hip-joint.
15. Resting with both hands on chair in front, raises foot by flexing knee without movement at hip.
16. Resting one hand on chair at side, patient swings opposite extended leg outward from hip-joint, then returns to normal.
17. Arms rotated outwards and inwards from shoulder-joint, operator grasping the metacarpal portion of the hand.
18. Wrist-joint flexed and extended.
19. Ankles dorsoflexed and extended alternately.

When these precautions are taken the exercises have an excellent effect in a considerable number of cases, bringing about relief of the dilatation and more or less immediate improvement (increased tonicity). An example of this is shown in Fig. 135, illustrating the diminution in the cardiac shadow under the X-ray after a very few resisted movements. On the other hand, there is the greatest danger that the treatment will be applied in cases where it could not have been expected to do good and where it actually does harm, producing overstrain and decreased tonicity of the cardiac muscle.

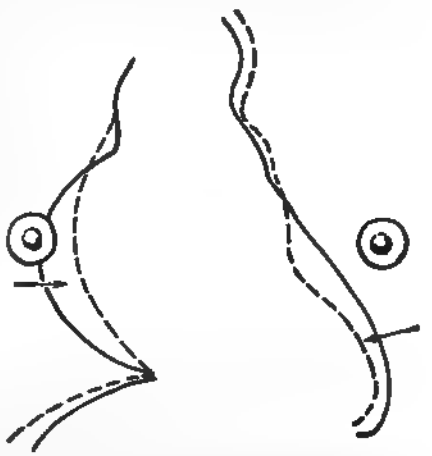

FIG. 135. — Orthodiagraphic outline of a patient with dilated heart, showing the effect of Schott movements. (After W. Bezly Thorne.) Solid line, outline before treatment; broken line, outline after resisted movements.

MECHANOGYMNASTICS.

Movements may also be carried out by means of the elaborate and ingenious apparatus devised by Zander for regulating them in direction and intensity. In these exercises the movements are semi-passive, being determined to a great extent and carried on by the apparatus. Hence it becomes more difficult to control them accurately than is the case with the resistance movements. It is unquestionable that excellent results have been obtained by this method, especially in cases where there is mild dilatation but no serious heart lesion; but it is certain that the limits of the patient's strength are too readily overstepped; and equally certain that, in the large institutions where this is carried out, the superintendents usually pay so little attention to the individual patient that these exercises very frequently do distinct harm.

WALKING AND CLIMBING.

The question of walking involves not only an important form of exercise treatment but also the regulation of the convalescent's daily life. As has been stated above, walking up and down stairs frequently introduces

the greatest strain upon the patient's heart. It is most important that this strain should be minimized. This may be done by causing him to rest upon each step long enough to count five, ten, or twenty, thus insuring him against hurry and breathlessness (J. O Hirschfelder). Another method which has been found useful was suggested by the writer's wife while climbing mountains in the Sierra Nevadas She noticed that she could climb quite steadily up the steepest trails provided she took a deep or normal inspiration each time the same foot touched the ground. In this way a relation was established between speed and respiration, the former was regulated by the latter, and a certain balance maintained between the rate at which oxygen was used up and that at which it was supplied. As the pulse-rate is often some definite multiple of the respiratory rate, this procedure also tends to regulate the former. This rhythm is one which is very satisfactory for patients with heart disease. It is readily acquired, and, having once become habitual, does much, automatically, to keep the patient within his physiological limits, thus enhancing the beneficial effect of the exercise while establishing a safeguard against overstrain.

Oertel's Mountain Climbing.—Long walks and mountain climbing were introduced as an after-treatment in cardiac disease by Oertel. Oertel found that patients convalescent from heart failure, and especially those suffering from fatty infiltration of the heart, were much benefited by long walks taken slowly, interrupted by frequent rests. Walks along gradually sloping paths in the mountains were most beneficial, and in fact became a feature of the method. This is designed, however, only to put the finishing touches upon the treatment, and to fit the patient whose heart is already in good working order for the more strenuous life to be pursued after his discharge.

CHOICE OF EXERCISE.

As regards the choice and use of exercise in treatment, the following general principles may be laid down:

(1) No exercise should be begun until the patient has been under observation for a few days, so that his general condition is thoroughly understood.

(2) If the patient is not improving under absolute rest, exercises would only increase the work imposed upon the heart and would do harm.

(3) If the patient has improved under absolute rest, he may be given one or two passive movements (each carried out five or ten times) two or three times a day, and the exercises very carefully increased in number and intensity each day before allowing him to get out of bed Even a few mild resisted arm exercises may be tried, bearing in mind the same principles, for it must be remembered that the patient may obtain much more complete and immediate rest after these exercises while in bed than when out of it, and also that he is not at the same time subjected to the strain of standing.[1]

[1] The relative mildness of such exercises in patients still bed-ridden is seen in the fact that their pulse-rate and respiration return at once to normal on cessation of the exercise. Physiologically, to exercise in the horizontal posture increases the systolic output more and changes the pulse-rate less than in the erect posture (Erlanger and Hooker).

Once out of bed the patient should at first be given a day or two of complete rest to accommodate himself to the new position. Then he may be allowed to begin gradually with a few of the resisted movements, if a competent attendant or physician can supervise them; if this is not available, he may be allowed to practise a few exercises in contracting antagonistic muscles (Selbsthemmungsbewegungen), at first under the direction of the physician, later under the observation of a skilled attendant, or of some reliable member of the family who has been carefully instructed in the precautions given above. About this stage the bath treatment may be begun.

(4) Mechanical gymnastics (with the Zander apparatus or modifications thereof) can be recommended only when supervised by persons of great experience and excellent judgment.

Training at End of Treatment.—(5) When the patient has recovered somewhat, but not sufficiently to withstand the wear and tear of daily life, he should be encouraged to take short walks, gradually lengthening the space covered, at first about the hospital grounds, later about the city or country, keeping records of the distance traversed each day. He may then be allowed to walk up hill. *Pari passu* with this the resisted or antagonized movements and the baths should be given. Before discharging the patient, he should be compelled to take some regular gymnastic exercises every day and made to do work at least as strenuous as that which will form the routine of his daily life after passing from under the physician's care. It is no more fair to the convalescent to put him directly back from the sedentary life of the bedroom or the hospital to the deadly struggle for existence outside than it would be to match the average citizen against a prize-fighter. He must be gradually trained for the effort. This principle was very well recognized by da Costa during the Civil War. Before sending his patients back to their regiments where they were subject to heavy field duty, forced marches, etc., he kept them at lighter duties about the hospital, upon local guard duty, etc., and from time to time during this period subjected them to tests of increasing severity (running races, etc.) until he was quite certain of their ability to stand the strain. The magnificent results which he reports from his large series of cases treated under otherwise unfavorable conditions constitute a fitting monument to one of America's greatest clinicians, and merit the careful study of all who would learn how cures should be obtained in heart diseases.

Treatment and Occupation.—On the other hand, the training to which the patient need be subjected should be suited to the life that he leads. It would be unnecessary to train a clerk in a store up to the point of muscular strength that is necessary for the ordinary laborer. But it is necessary that he should not be exhausted by a few hours' standing lest the cardiac overstrain return. On the other hand, when *restitutio ad integrum* has not been possible, the patient's life must not be the same as it was before his illness. His work must be cut down. This may often be done in the more well-to-do without changing the business by employing assistants to attend to all except the more essential affairs. Poorer persons must change their occupations. It is as much the duty of the physician to see that this is done after the recovery as it was his duty during the height of the illness to give correct treat-

ment. Otherwise he has merely prepared the patient for another break-down. The difficulty in finding suitable occupation and the acumen necessary in meeting changed conditions increase rather than decrease the responsibility of the physician in this regard. He must see to it that, as stated by Professor Osler, "the patient must always live within his income of cardiac energy." His mode of life, and especially the speed of his movements and the intensity of his efforts, should be so regulated that he no longer feels at any time palpitation, shortness of breath, or precordial pain.

HYDROTHERAPY IN THE TREATMENT OF HEART DISEASES.

Although the healing power of mineral springs and baths was thought by the older physicians to be well-nigh universal, the scientific application of hydrotherapy to heart disease is due largely to the studies of a small group of men at Bad Nauheim, Germany. Benecke, in 1870 noted the favorable action of baths at this watering-place, but it is to August Schott that is due the real credit for introducing into cardiac therapy what is really a very valuable method of treatment.

PHYSIOLOGICAL ACTION OF BATHS.

Physiologically it has been found, especially by Erlanger and Hooker, and a little later by Jacob and Strasburger, that all baths given at about the temperature at which the body neither gives off nor loses heat (92° F., 33° C.) increase the pulse-pressure and slow the pulse-rate. Strasburger found this to be particularly true as regards baths of the same composition as those at Nauheim, or indeed any other baths in which CO_2 is effervescing; and ascribes this action to the dilatation of the vessels in the skin over the whole body, as well as to the cardiac reflexes from stimulation of the sensory nerves by the prickling sensation of the CO_2. These effects in themselves would be sufficient upon *a priori* grounds to indicate a probable value of such baths in weakened hearts. Schott's treatment has, however, long antedated these explanations. Schott, Thorne, Schmanke, and a host of other observers have demonstrated that the area of cardiac dulness and the X-ray shadow of the heart diminished after such a bath (cardiac tonicity increased).

An excellent treatise of his results and those obtained by other observers is given *in extenso* in English in the monograph of W. Bezly Thorne, to which the reader is referred for details of the method. Other excellent accounts are given by Satterthwaite, P. K. Brown, et al.

PRECAUTIONS.

The baths should not be given to patients who are in the extreme stages of cardiac break-down, nor indeed to any very weak patients, until they have been prepared for the slight strain which accompanies them by some course of mild exercises, preferably resistance exercises (see page 195). They should never be taken less than one or two hours after a light meal or four to five hours after a heavy one, and, on the other hand, should not be given upon an absolutely empty stomach.

NATURAL AND ARTIFICIAL NAUHEIM BATHS.

The Nauheim baths are obtained from several mineral springs of different composition. A course of baths is begun in the Great Spindel composition H_2O 1000, NaCl 2.18, KCl 0.5, $CaCl_2$ 1.7, $MgCl_2$ 0.4, calcium bicarbonate 2.3, CO_2 3.17, temperature 31.6° C., 88.8° F.), most of the CO_2 being allowed to escape before immersion of the patient.

The effect of the Nauheim baths can be imitated at home or in the hospital by adding the same salts to the water in the bath-tub. A great variety of such artificial Nauheim salts are on the market, put up in packages ready for use. The most satisfactory known to the writer contains:

	Grammes	Pounds	Per cent
Sodium chloride	4500	8	2.2
Calcium chloride (magnesium chloride)	900	2	0.53
Sodium bicarbonate	800	1¾	0.1
Sodium bisulphate yielding CO_2	1000	2¼	0.29

In order to prevent the bisulphate from injuring the tub it is advisable to cover the walls and floor of the latter with a large sheet of rubber cloth about 6 x 8 ft. in size. The bath is filled with warm water, 90°-95° F., (a good-sized bath requires 40 to 45 gals. 150 to 175 litres) and the salts added—first the sodium chloride, then the calcium chloride, then the sodium bicarbonate and lastly the acid sulphate ($NaHCO_3 + NaHSO_4 = Na_2SO_4 + CO_2 + H_2O$). The effervescence continues throughout the bath.

CAUTIONS IN GIVING BATHS.

In preparing the first bath it is better to begin with half strength of the salts or even less. The patient is allowed to remain in this bath not longer than fifteen minutes, being watched carefully during this time and removed at once if there is the slightest increase in cyanosis or real discomfort of any kind—flushing, excitement, or syncope. "The immediate effect of the first few baths is to produce a sense of oppression at the precordium, under the influence of which the patient breathes slowly and deeply for two or three minutes. Respiration then becomes easy and continues slower by from two to four breaths a minute," after which the symptoms subside. In general the effect should be similar to that in the following case quoted from Thorne:

"A patient, aged 46, whose health had been declining for years, was found to have a pulse of 80 in the recumbent, and of 88 in the sitting, position. While he stood it varied from 100 to 104, and if he walked ten paces it rose from 120 to 130. The apex was found to beat an inch outside the nipple line. Within two minutes of immersion in his first thermal bath the pulse had fallen to 70, and judged by the finger appeared to have doubled its volume,[2] at the end of four minutes it was 68, in six minutes 66, in eight minutes 68, and while standing after the bath it was 90. Before he left the bath after an immersion of ten minutes, the apex beat was found to have receded half an inch in the direction of the mesial line, and nails and fingers, which had been snow-white up to the junction of the second with the first phalanx, had assumed a healthy flesh tint."

This healthy reaction of the skin should be present within a few minutes after the bath. Its absence indicates that the treatment has been too

[1] Put up by R. R. Rogers Chemical Co., San Francisco. This preparation is particularly useful, owing to the excellent grade of sodium bisulphate prepared and the permanent and convenient form in which it is put up. Moreover, the sodium bisulphate is put up in lumps the size of a hazel-nut, which allows the CO_2 to be generated uniformly throughout the bath.

[2] Probably the pulse-pressure had actually doubled.

violent, too prolonged, or in other ways unsatisfactory, and unless this can be obviated after the next bath or two the treatment should be discontinued.

After the bath the patient should be made to lie down and rest, if possible to sleep, for at least an hour before leaving the building or doing anything else, and upon this rest as much as anything else depends the success of the treatment.

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VII.

HYPERTROPHY AND ATROPHY.

HYPERTROPHY.

To enable the heart to recover from an overstrain and the consequent dilatation, to maintain the circulation in the presence of a valvular lesion or dilatation, or to reestablish compensation once broken, it must put forth an increase in force. The stimulus for this seems to lie in the increase in residual blood in the ventricle, which acts as an increase in load upon the heart muscle, and thus tends to increase both irritability and force of contraction, as shown by O. Frank (see page 135), and particularly to bring

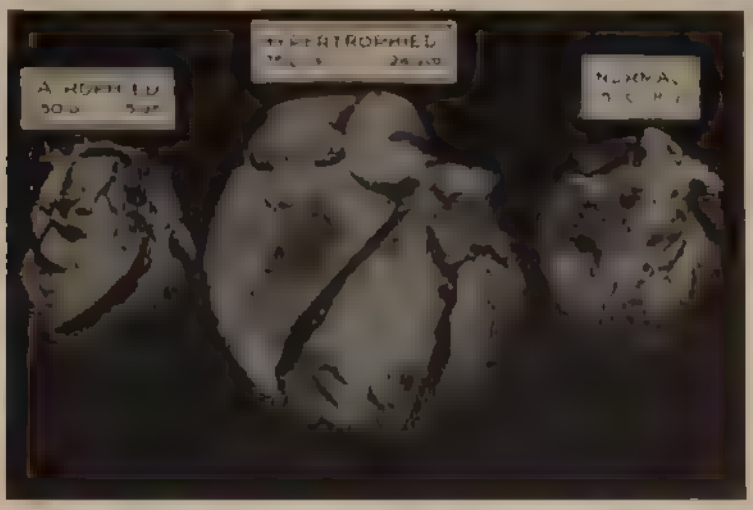

FIG. 136. Hypertrophic, normal and atrophic hearts. From specimens in the Army Medical Museum, Washington, D. C.

about an increase in tonicity. It seems probable that this increase in tonicity is of primary importance as a predisposing factor to hypertrophy, and Barcroft and Dixon have shown that increased tonicity is accompanied by an increased CO_2 metabolism in the heart.

PATHOLOGICAL ANATOMY.

Changes in the Fibres. The main visible change which the heart muscle undergoes is a swelling of the individual fibres (Tangl, Goldenberg, Delafield, R. M. Pearce) with little if any multiplication of the muscle-cells. Goldenberg finds that the muscle cells in the wall of the hypertrophic heart have a diameter of 17.65 μ, in the normal heart 12.85 μ, and in the atrophic heart 10.84 μ. The striation of the fibres also becomes less distinct, and vacuoles appear in the sarcoplasm, changes which are similar to what is observed in

a striated muscle as the result of prolonged contraction. Ranke has shown that in skeletal muscle these changes are due to imbibition or endosmosis of water, which, according to the beautiful experiments of J. Loeb and his pupil, Miss Cooke, is brought about in the following way. During the muscular contraction the more complex molecules break down into several simpler ones, thereby increasing the number of molecules in solution in the muscle plasma; the osmotic pressure rises, and hence brings about an endosmosis of water into the fibres. Having once entered, the water molecules remain and the muscle swells. There can be little doubt that the same process is going on in cardiac muscle, especially when subjected to overwork, but no observations have actually been made upon this phase of the subject.[1]

In cardiac hypertrophy three anatomical changes may be said to take place simultaneously: (1) an increase in size of the individual muscle-cells, but apparently no increase in their number; (2) a certain amount of

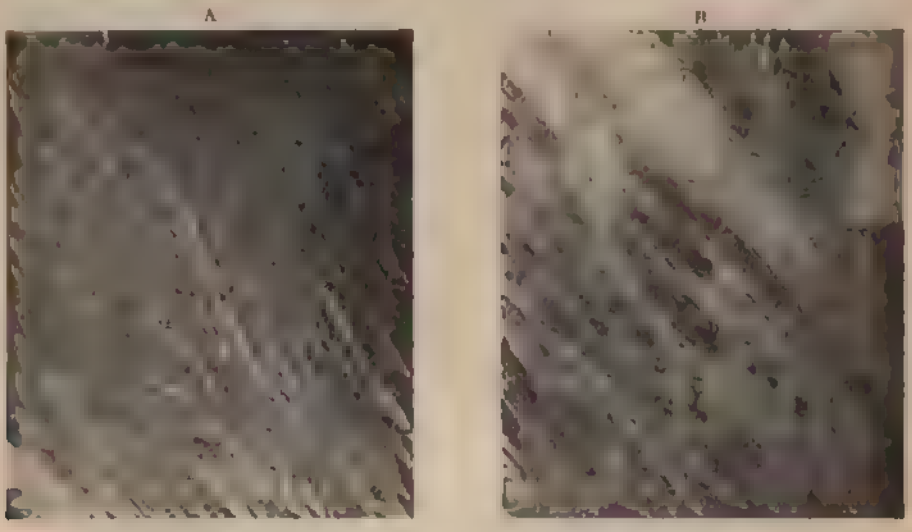

FIG. 137. Photomicrographs of atrophic and hypertrophic heart muscle. A, Atrophic heart muscle, showing stone cells. (The specimen also showed some oedema and slight mononuclear infiltration between the muscle-cells.) B, Hypertrophied heart muscle, showing large cells with swollen nuclei.

degeneration is almost always present in some of the muscle-cells; (3) a proliferation of the strands of connective tissue between the bundles of muscle-fibres (interfascicular myofibrosis, see page 234).

Dehio and Pearce have shown that each fibre may pass through the following stages: normal → hypertrophy → degeneration, the latter stage being associated with proliferation of interstitial connective tissue (myofibrosis). Accordingly, we may find the heart-cells in the following conditions:

(1) Normal + hypertrophied (heart somewhat enlarged, as in athletes, also in Kulbs's dogs).

(2) Hypertrophied + degenerated, some proliferation of connective tissue (heart much enlarged—cor bovinum—still strong).

(3) Degenerated. Marked proliferation of connective tissue. Marked weakness of the heart. Large failing heart. Hypertrophy + dilatation (digitalis often harmful).

[1] Heseher and Leo Loeb have advanced the same explanation.

Types of Hypertrophy.—Hypertrophy was supposed by Cohnheim to assume three types:

(1) General concentric hypertrophy, involving all the chambers of the heart about equally.

(2) Local concentric hypertrophy, involving the walls of one or more chambers of the heart which is subjected to extra work. The fibres are not especially elongated.

(3) Local (excentric) hypertrophy with elongation of the muscle-fibres, as in aortic insufficiency. The elongation of the fibres is somewhat out of proportion to the increase in size of the heart.

The existence of these three types of hypertrophy as separate entities was already disputed by Cruveilhier in 1833. It is probable that the size of the cavities as found at autopsy bears no constant relation to that present during life. Moreover, the ventricular cavities in cases of chronic nephritis are often quite as large as those in hearts of aortic insufficiency, though the former typifies the so-called concentric, the latter the excentric hypertrophy.

Occurrence and Sites of Hypertrophy.—The relative frequency with which these factors occur in cases of hypertrophy is shown in the following statistics compiled by W. T. Howard from autopsies made in the Pathological Department of the Johns Hopkins Medical School upon 108 subjects showing hypertrophy of the heart.

	Cases.	Per cent.
Arteriosclerosis	65	59
Nephritis	14	13 1
Valvular lesions of the heart	13	12 1
Adherent pericardium	8	7 6
Hard work	4	3 8
Tumors	2	1 9
Aneurism of the heart wall	1	0 95
Hæmic plethora	1	0 95
Total	108	100

The right ventricle showed hypertrophy in 70 cases (66 per cent.), of which there were—

Arteriosclerosis (often of pulmonary artery), 52; adhesive pericarditis, 6; valvular lesions, 8; chronic nephritis, 3; hydræmic plethora, 1.

Hypertrophy of the auricles (atria) was most marked in mitral stenosis and adhesive pericarditis.

Strain, Exercise, and Hypertrophy. In normal individuals the weight of the heart is almost proportional to the weight, not of the entire body, but of the musculature (W. Müller, Hirsch), being relatively low in fatty and relatively high in muscular individuals. The absolute weight of the heart is about $\frac{1}{250}$ (.0059) of the body weight in men, $\frac{1}{183}$ (.00546) in women. The same general principle applies in animals, the most active animals having the largest hearts, especially race-horses, hares, etc., as compared to less active members of the same species.

When, however, the heart is subjected to abnormal strain, especially as the result of valvular lesion, it hypertrophies and increases in size to dimensions which are often enormous. It is not very uncommon to find

hearts of twice or even three times the normal size (500 to 800 Gm., 17 to 26 oz.), and in the Army Medical Museum in Washington there is a specimen of one weighing 1000 Gm. (33 oz.). Another heart of 1400 Gm. (46½ oz.) has been reported. Such a heart is usually designated as a b e e f y h e a r t or *cor bovinum*, indicating the animal to which its size would be proportioned.

Work Hypertrophy.—Whether a true hypertrophy occurs in a perfectly healthy heart has been much disputed, many writers taking the stand with Romberg that, "though the possibility of a 'work hypertrophy' cannot be denied, more proofs of its existence are necessary."

FIG. 138.—Heart (A) of normal dog and (B) of dog which has run for three months on a tread-mill. (After Kulbs, *Arch. f. exper. Path. u. Pharmacol.*, lv.)

Recently, however, absolute proof of a work hypertrophy without myocardial degeneration has been brought by the beautiful experiments of Kulbs. This observer took two dogs of the same litter and of equal size, kept them in neighboring cages upon the same diet, but compelled one of them to run upon a tread-mill daily for three to six months, while the other was kept quiet and used as a control. At the end of this time both dogs were killed in the same manner.

	First set		Second set	
	Work-dog	Control	Work-dog	Control.
Total weight	15,200	15,000	19,200	20,400
Musculature	5,696	5,342	6,489	6,776
Heart	152	99	172	113

Kulbs's results have been confirmed by Grober and by Joseph.

The increase in size of the heart was not accompanied by any change in the skeletal musculature, nor were any pathological changes present in the heart or arteries. The muscle here simply underwent an increase in size, the purest form of hypertrophy. Kulbs's dogs were simply in training to run on a tread-mill. The process was exactly the same as the "training" of an athlete, and clinically, it is often found that athletes have mildly hypertrophied hearts. Schneffer has demonstrated with the orthodiagraph that the size of the heart is increased in persons whose occupations require hard work and decreased in those with sedentary callings. He has also shown that the hearts of the young men doing military service in the German Army increase in size somewhat during their period of service. However, these men often indulge in excess of alcohol or tobacco, so that before they die enough myocardial change has set in to justify the scepticism of men like Krehl (l. c.) and Romberg (l. c.). Nevertheless, though a true "work hypertrophy" must be admitted, in these experiments it is noticeable that the increase in weight of the heart

amounted to only 52 per cent as compared with changes of 100 to 300 per cent. often observed in man It is doubtful whether a corresponding degree of hypertrophy would be noticeable clinically.

ETIOLOGICAL FACTORS.

Hypertrophy in Chronic Nephritis.—The most remarkable and most important of all these forms of hypertrophy is that taking place in chronic nephritis. This was first noticed by Richard Bright in his classical description of dropsy in nephritis.

In 1853, Wilkes thought that the lesions of the kidneys and arteries were part of the same morbid condition, while Gull and Sutton assumed that the general arteriocapillary fibrosis brought about an increased resistance through narrowing of the arterial bed, and, as a result of this, high blood-pressure and hypertrophy of the heart Senator ascribed the hypertrophy to a "dyscrasic" property of the blood in nephritis, stimulating the heart to contractions of abnormal force. Pässler and Heineke have recently subjected the matter to critical experiment They found that if they cut out pieces of kidney from a dog bit by bit until renal substance equal to 1¼ kidneys had been removed, the heart then began to hypertrophy and the blood pressure to rise If considerably more tissue was removed, the animal became cachectic, the blood-pressure remained low, and the heart did not hypertrophy They ascribed these cardiac changes, as George Johnson had done, to the presence in the blood of some substance having a digitalis-like action, being either retained in the circulation in abnormally large quantities as the result of disturbed excretion, or being a true internal secretion from the diseased kidney[1]

Numerous other theories of cardiac hypertrophy in renal disease have been advanced Chief among these is the theory of J. Cohnheim and Traube that the sclerosis of renal vessels narrowed the arterial bed in the kidney, thereby introducing an increased resistance into the general circulation, and that these changes in the renal vessels were enough to raise the general blood pressure. It would appear in the light of more modern research that this cutting off of the blood stream is in itself insufficient On the other hand, Buhl, Huchard, and Albrecht have suggested that the hypertrophy is not a true one but simply a pseudohypertrophy (interfascicular myofibrosis, see page 234), the entire increase in size of the heart being due to growth of connective tissue and not of the heart muscle, but histological examinations do not bear out this view.

Hypertrophy from Overdrinking.—Closely allied to this condition is the tremendous heart hypertrophy which is universally found to result from drinking large quantities of beer, and, since it does not accompany excess in any other form of alcohol to the same extent, it is thought to be due to the large quantity of fluid ingested That increase in the fluid in the blood at once results, not so much in a rise in arterial blood-pressure as in rise in venous blood-pressure, dilatation of the heart, and increases in the systolic output, even to the point of doubling or trebling it, can easily be shown with Henderson's cardiometer, and this no doubt illustrates the mechanism by which the change is brought about.

Hypertrophy and Arteriosclerosis.—The relation of hypertrophy of the heart to arteriosclerosis independent of any renal changes is also of fundamental importance. The coincidence of the two conditions in the same individual has long been noted, and both have been brought about experimentally by administration of certain poisons, notably adrenalin (Josué, Erb, Pearce, et al.).

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[1] Tigerstedt and Bergmann (Skand Arch. f Physiol., Leipz., 1898, viii, 224) found that injection of renal extract actually raised the blood-pressure, owing to the presence of a substance which they named "renin."

Cardiac and Adrenal Hypertrophy.—A new light has been thrown upon the subject by the studies of Vaquez and Aubertin (1905), Aubertin and Clinet, Wiesel, and Gaillard.

Aubertin was able to produce cardiac hypertrophy in rabbits by various means, and found in every case a simultaneous hyperplasia of the medullary substance in the adrenals. A similar finding had been made by Vaquez and Aubertin in cases of chronic nephritis associated with hypertrophy of the left ventricle, which was confirmed by Wiesel in 1907. In December, 1907, Aubertin and Clunet made a study of 120 unselected autopsy cases. Of these 18 showed very definite hypertrophy of the medulla of the adrenals, and 16 of these 18 showed marked hypertrophy of the heart. On the other hand, but 10 of these hypertrophied hearts were associated with renal disease; the others occurred in conjunction with valvular lesions, congenital defect in the septum ventriculorum, aortic sclerosis, etc. Aubertin, however, states very definitely that besides these groups they encountered cases of cardiac hypertrophy without the existence of adrenal hyperplasia, so that this association is not invariable; and they conclude that it is at present impossible to decide whether the cardiac hypertrophy occurs as a result of oversecretion of adrenalin, or whether the hyperplasia of the adrenals occurs as a result of slight venous stasis in these organs while the hypertrophy is going on. Arteriosclerosis was the rule but not invariably in these cases with adrenal hypertrophy.

It must be noted that the action of adrenalin is just that which might be expected to bring about hypertrophy of the heart, for it causes, (1) a general vasoconstriction; (2) a marked increase in the tonicity of the heart, (3) an increase in the force of the beat and in the systolic output.

However, the results of Cohn, under Aschoff's direction, are less favorable to this theory. In 12 cases of hypertrophy of the left ventricle with chronic nephritis, he found hypertrophy of the adrenal cortex in only 3 (25 per cent.), while in 23 cases of chronic nephritis without hypertrophy of the heart he found hypertrophy of the adrenal cortex in 8 (34 per cent.). These findings tend to throw considerable doubt upon the theory of Vaquez and Wiesel.

Hypertrophy and Abdominal Arteriosclerosis. Hasenfeld has found that no hypertrophy sets in unless arteriosclerosis is present in the aorta above the level of the superior mesenteric artery. Practically all the substances which are known to bring on arteriosclerosis are vasoconstrictors, and beginning arteriosclerosis in man seems usually to be accompanied by vasoconstriction. It is readily conceivable that any sclerotic obstruction below the mesenteric would be easily compensated for by dilatation of the abdominal vessels, and, consequently, would bring about no increased resistance to blood flow, while at the higher level the presence of sclerosis is more or less equivalent to clamping the abdominal aorta.[1]

DIAGNOSIS.

It would appear at first sight to be extremely easy to determine clinically whether in a given case hypertrophy is present or not, and the older clinicians laid down very definite rules for its detection, most of which were fallacious. In general, we may agree with Gibson that the most important signs of hypertrophy of the left ventricle are increase in cardiac dulness to the left, with a more or less steady, forceful, and "heaving" impulse, and a

[1] An excellent discussion of the theoretical and experimental side of the question is given by R. M. Pearce.

booming first sound of low pitch, and an accentuated second sound at apex and aortic area. These signs are dependent largely upon the contact of the heart with the chest wall; and if, as is often the case in an emphysematous individual, the lung intervenes between the left border of the heart and the chest wall, all the signs may be diminished beyond recognition. The diagnosis may, however, often be made from the history in spite of the clinical findings. Thus, if an aortic or mitral insufficiency has persisted for some time and the heart is in a condition of moderate vigor with a normal pulse-rate, it may be assumed that hypertrophy of the heart has had to take place in order to maintain the circulation, in spite of distant heart sounds and absence of the apex beat. Prolonged high blood-pressure is usually associated with some degree of hypertrophy of the left heart, but not invariably. In differentiating from dilatation it may be stated that, except under unusual conditions brought on by stimulation of the vagus, the factors bringing on dilatation quicken the pulse-rate, and an enlarged but slowly beating heart is almost always hypertrophied. In hypertrophy of the left ventricle, in contradistinction to that of the right, the maximum impulse is usually a systolic protrusion, while in the latter case it is a systolic retraction. The latter is also frequently the case when both ventricles are hypertrophied.

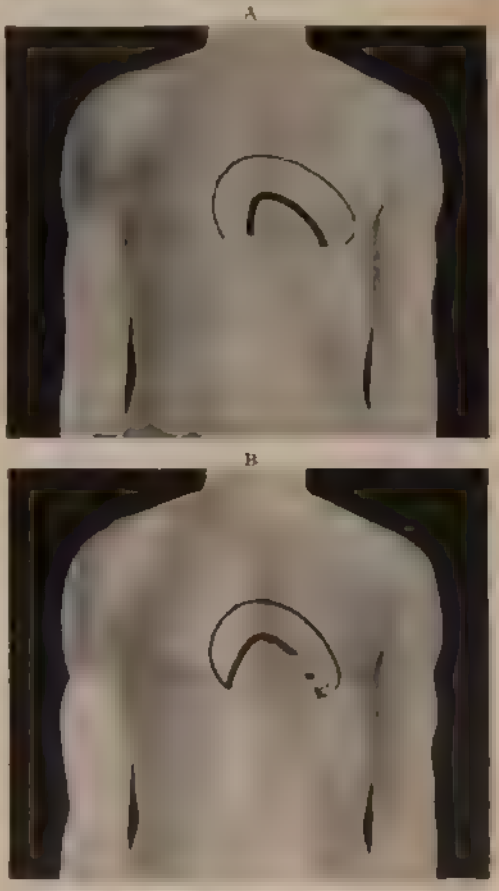

FIG. 130.—Areas of pulsation and retraction hypertrophy of the right and left ventricles. [illegible] retraction, [illegible] pulsation. The [illegible] the area of the cardiac dulness. A. Hypertrophy of the left ventricle. B. Hypertrophy of the right ventricle.

Hypertrophy of the Left Ventricle.—Palpation of the apex impulse, which many writers, even as late as Romberg, consider a most important sign of hypertrophy of the left ventricle, need not be decisive, since, as Katzenstein has shown, the weakest hearts may often beat the most violently, especially when beating rapidly; the strongest, on the other hand, may be separated from the chest wall by a layer of lung. Dulness is, however, increased to the left.

Hypertrophy of the Right Ventricle. The hypertrophy of the right ventricle is not so easy to diagnose. Its presence may be inferred when the area of cardiac dulness is enlarged and a systolic retraction is

noted at the point of maximal impulse and over the interspaces between it and the sternum as well as in the epigastrium. The heart need not be enlarged toward the right, since the right ventricle rarely passes the sternal margin. Indeed it rather tends to lift the apex and shift it to the left. The area of cardiac flatness is increased to the right, reaching to the sternal margin. An increased area of dulness to the right of the sternum is due to the right auricle. The second pulmonic sound is intensified and ringing, but this may also be the case in any condition in which there is some obstruction to the pulmonary circulation or some insufficiency of the left heart.

Hypertrophy of the auricles cannot be diagnosed from objective signs except in mitral stenosis, in which an hypertrophied auricle gives rise to a loud presystolic murmur. This is not present when the auricle is weak. Hypertrophy of the right auricle is sometimes shown by a high presystolic wave upon the jugular venous pulse-curve and very rarely by a presystolic wave upon the liver pulse (Mackenzie); but, as a rule, it shows no signs.

Prognosis.—A certain amount of hypertrophy is necessary whenever a valvular lesion or any other abnormal factor tending to increase the work of maintaining the circulation is present. Hence failure of the heart to hypertrophy under these conditions would be regarded as an unfavorable condition, and would probably soon be associated with cachexia. On the other hand, an extreme degree of hypertrophy is evidence that the heart is doing its maximal work, that the fibres ere long will begin to degenerate, and the heart must be spared as much as possible.

Hypertrophy in itself does not demand treatment, but diminution of the causal factor as far as is possible is advisable. If this be nephritis or arteriosclerosis, a quiet life and diet poor in salt and purin bodies should be resorted to, with occasional courses of potassium iodide. If a valvular lesion be present and the hypertrophy is slight, little attention need be paid to it until the patient reaches the latter half of the fourth decade, when he should begin to spare his heart and arteries as much as possible, should abstain from alcohol, coffee, and tobacco, and should in every way avoid those influences leading to the production of high blood-pressure and arteriosclerosis.

Reserve Force of the Hypertrophied Heart.—One of the most important questions that arise in connection with hypertrophied hearts is whether or not a hypertrophied heart possesses as much reserve force as a normal one. This question is variously answered in the text-books, most of them agreeing with Krehl et al. that the reserve force is lessened, while the experimental work, especially that of Romberg and Hasenfeld, indicates that the strength of the hypertrophied heart muscle itself is actually increased. However, a great deal depends upon the stage of hypertrophy in which the individual heart happens to be. Thus a heart in the first stage, with fibres normal and hypertrophied, would show an increased strength (as in athletes' hearts, or in hearts of early hypertrophy after valvular lesion as compared to the same hearts at the very onset of the lesion), while a heart in the second stage, with fibres partly hypertrophic, partly atrophic, would in most cases show a marked diminution in

strength and still greater loss in reserve force, and an increased effort would hasten the degeneration.

Another and really main factor in the apparent weakness of the hypertrophied heart is that in practically all hearts the hypertrophy is brought on by some valvular lesion or by some persistent increase in peripheral resistance; so that such hearts are continually wasting much of their energy in overcoming these pathological conditions, besides bestowing the usual amount of it upon the maintenance of the circulation. In bodily exertion or other conditions calling upon the reserve force, not only the actual circulation must be increased, but the abnormal factor inducing wasting of energy, the valvular lesion, etc., becomes more severe as well, and hence the extra call upon the diseased heart is double the extra call upon the normal and requires double the reserve force to meet it. Otherwise the reserve force, though actually more, may be apparently less than in the normal heart, as shown diagrammatically in Fig. 140. For practical purposes, however, it may be regarded as indisputable that, in every case where a cardiac lesion is present, the hypertrophied heart has less available reserve force than normally, and in some cases (stage 3) less than if it had not hypertrophied at all.

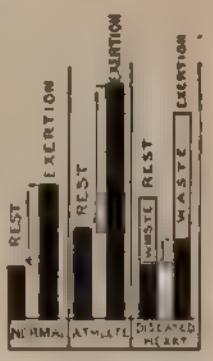

FIG. 140. Diagram showing power of normal and hypertrophied (athlete's) heart at rest and during exercise, also that of a diseased heart. The length of the arrow indicates the reserve force. The unshaded portion indicates the cardiac energy expended but wasted, owing to the lesion.

ATROPHY.

Atrophy of the heart is more or less the reverse process of hypertrophy. Whenever the body diminishes in weight from cachexia, infectious disease, or starvation, the heart muscle diminishes with it, and according to Hirsch in about the same ratio. The epicardial fat, on the other hand, is but little diminished. When the atrophy is the result of starvation it may be of very high degree, but the size and condition of the heart may return to normal when an adequate diet is resumed (Schieffer).

As in the case of hypertrophy, there seems to be little change in the number of the muscle-cells, but the latter diminish in size (10.84 μ instead of 12.85 μ, Goldenberg), and the removal of substance is marked by the deposition of brown granules of hæmatoidin in fusiform arrangement about the nucleus. These granules are formed when part of the muscle-cell proteid is broken down during the atrophy, the hæmatoidin portion being left. Macroscopically they impart a tobacco-brown color to the heart, so that the condition is often designated as "brown atrophy of the heart."

To a certain extent a diminution in size of any chamber of the heart may occur if its work is lessened by obstruction to the blood flowing into it; as, for example, the left ventricle in pure uncomplicated mitral stenosis. The atrophy is rarely so marked here as in starvation, phthisis, or cachexia, and is indeed the exception rather than the rule in mitral stenosis, for other factors, tachycardia, irregularity, or mitral insufficiency, usually contribute to keep the left ventricle doing an at least normal amount of work.

Like hypertrophy, cachexial atrophy of the fibres may lead on to growth of interstitial connective tissue and fibrous myocarditis, but true brown atrophy is not so common a forerunner of myocarditis as is hypertrophy of the heart. Functionally, the force of the heart is impaired about proportionally to its diminution in weight. The blood-pressure is usually low and the muscle easily fatigued. Overstrain readily occurs in such hearts; and sudden death is not uncommon.

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VIII.

FATTY DEPOSITS IN AND ABOUT THE HEART.

Fat may be deposited in the heart in two ways: (1) In solid masses of adipose tissue, especially in the pericardium in fat individuals, particularly in those addicted to alcohol, and very often associated with coronary sclerosis. This condition is designated as fatty infiltration or obese

FIG. 141.—Distribution of fat in and about the heart. *A*, normal, *B*, deposit in an obese heart, *C*, deposit in a fatty degenerated heart.

heart (Mastfettherz, Kisch). (2) In fine droplets occurring diffusely within the heart muscle-cells, especially in anæmia, infectious diseases, in persons poisoned by phosphorus, arsenic, and numerous other substances, and in association with other changes in the myocardium. This condition is called fatty degeneration.

FATTY INFILTRATION OR OBESITY OF THE HEART.

DEPOSITION OF THE FAT.

Harvey, the discoverer of the circulation, describes the hearts of certain fat persons as covered with a layer of fat so extensive as almost to obscure the heart muscle from view, and this condition is one of not very infrequent occurrence. In normal hearts there is a considerable amount of fat (30 to 60 Gm., 1 to 2 ounces) collected just beneath the endothelial layer of the pericardium, along the auriculoventricular and interventricular grooves (coronary and longitudinal sulci), at the base of the aorta, and scattered elsewhere over the heart. As the individual lays on more body fat, more fat is deposited in the pericardium, at first only at the usual sites along the sulci; but later it spreads over and into the myocardium, penetrating into it between the larger strands of muscle, and finally settling beneath the endocardium, especially about the bases of the papillary muscles.

The weight of adipose tissue may actually exceed the weight of cardiac muscle (W. Müller, Hirsch, Kisch), as shown by the following figures determined by W. Müller (for the method see page 4)

Total weight of heart	Heart muscle	Fat removable by dissection	Per cent. of fat	
253.6	240.7	12 9	5.1	Normal male (thin).
363 5	326 2	37.3	10 3	Cardiac hypertrophy.
327.6	181.3	146 3	45 6	Female } fatty heart.
494.3	228.3	266	53 5	Male }

Nature of the Fatty Deposit.—Under these circumstances the fat is deposited in exactly the same manner as elsewhere in the body. The pathological character consists not in the process but in the amount of the deposit. The adipose tissue in this region does not differ macroscopically or microscopically from the fat elsewhere. Neither does it differ chemically. It is ordinary "translocation fat" (Rosenfeld, Leick and

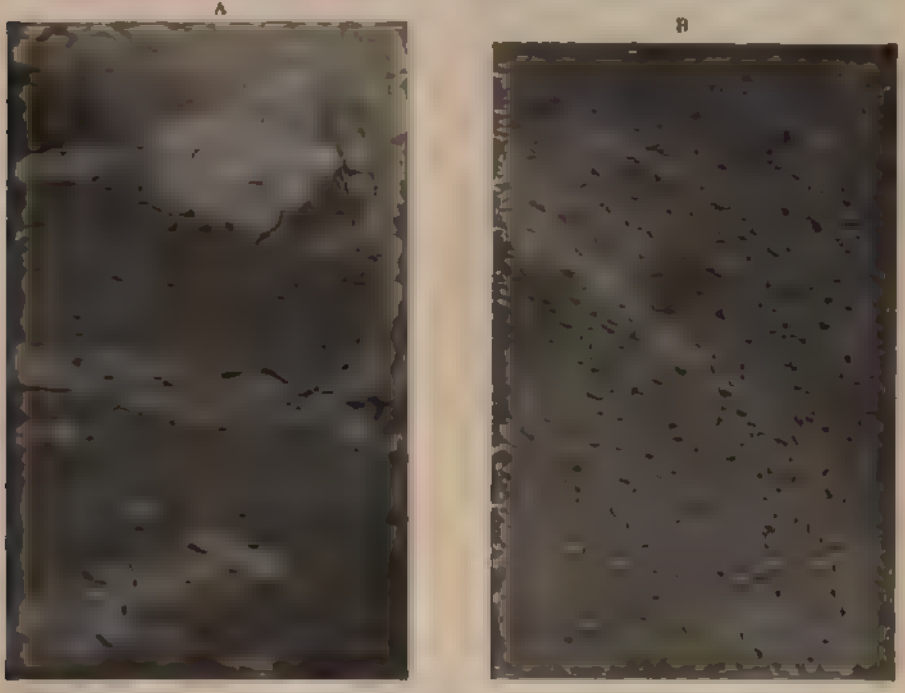

FIG. 142.—Photomicrographs of fat deposits in the heart. A, Heart muscle of an obese individual showing fat cells. B, Heart muscle of a patient who died of pneumonia, showing fat droplets within the cells; fatty degeneration. (Photomicrographs by Dr. Chas. S. Bond.)

Winckler, derived directly from the food; for Leick and Winckler have shown that if dogs be overfed with mutton tallow, the fat deposited in the pericardium has an iodine absorption coefficient approaching more nearly to what is in the sheep than to that of the dog. The pericardial fat differs from the fat elsewhere in but one important respect, and one which is especially to be borne in mind in treating the condition, namely, that it is relatively poor in lipase, the enzyme which forms and splits fat, and hence is relatively stable. According to Loevenhart it would appear that lipase is present in the cells in considerable amounts at the time the fat is deposited, but is then gradually destroyed, so that if subsequently the fat of the body is reduced from inanition or other cause, there is no more enzyme remaining in the pericardium to split up what is stored there and to return it to the general circulation. Accordingly, it is found that in starvation the pericardial and perirenal fat remain after all the rest has disappeared from the body (Loevenhart, Schieffer). This matter will be referred to again in connection with treatment.

CARDIAC CONDITIONS ASSOCIATED WITH OBESITY.

There are three definite conditions which, though in no way part of the general process of obesity, are often associated with it; and it is these, rather than the obesity itself, which give rise to the symptom complex referred to as "fatty heart," or, as Romberg more properly designates it, "cardiac insufficiency of fat persons" (Die Herzmuskelinsufficienz der Fettleibigen). These are (1) atrophy of the heart muscle, and (2) sclerosis of the coronary arteries, (3) a high diaphragm.

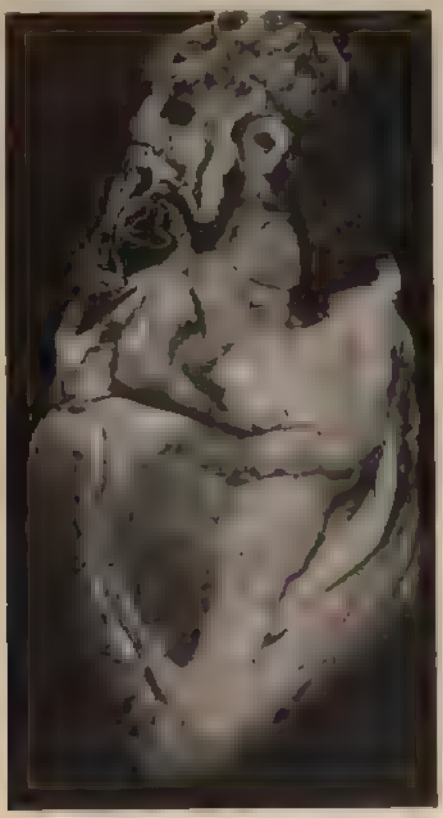

FIG. 143.—An excessive deposit of epicardial fat. (From a specimen in the Army Medical Museum, Washington, D. C.)

1. **Atrophy and Cardiosclerosis.**—It is especially worthy of notice that the increase in size and weight of the heart may conceal an actual atrophy of the heart muscle (Hirsch) (see table above) and a corresponding weakness of the heart. According to most writers, this lies mainly in the left ventricle, but Hirsch has shown that not only does the fatty **infiltration penetrate chiefly** the wall of the right ventricle, but that the symptoms most common among fat persons are those due to **primary failure of the right** side of the heart. A general cardiosclerosis (see page 235) is often associated with the deposit of fat. It is a self-evident fact that such enormous deposits of fat increase the work done by the heart, first by increasing the weight to be moved at each systole, and secondly by increasing the total bed of the blood stream. It might be supposed that this would in itself bring about hypertrophy, but hypertrophy is rarely demonstrable. The tendency to obesity usually occurs either in persons whose lives are sedentary and whose skeletal and cardiac muscles are therefore underdeveloped, or else in those addicted to excesses of alcohol or overeating, factors which in themselves bring on myocardial changes and hypertrophy.

2. **Coronary Sclerosis.** The pathological changes and symptoms due to sclerosis of the coronary arteries do not differ from those arising without the presence of abnormal fat deposits and will be discussed in a separate chapter (page 280).

3. **High Diaphragm.**—V. Frey and Krehl have shown in animals that pushing up the diaphragm, and thus displacing the heart, greatly inter-

feres with the work of the latter. Myers and Schott found that soldiers whose diaphragms are pushed up by tight belts about the abdomen exhibit symptoms of cardiac overstrain much more readily than do normal individuals. Myers found acute dilatations most common in the British regiments in which cuirasses and tight belts were worn.

Wenckebach has called attention to the fact that a large amount of intra-abdominal fat pushes up the diaphragm and thus pushes the heart into a more transverse position (apex often in the fourth interspace), thereby hampering its action. This factor must be reckoned with in the genesis of the cardiac weakness of fat persons.

ETIOLOGY.

Clinically, the cardiac manifestations in fat persons are very variable. They occur most frequently in association with (1) general obesity, either hereditary or arising primarily from over-eating; (2) in childhood; (3) after castration or menopause; (4) overindulgence in alcohol, especially malt liquors, with or without the presence of gout; (5) diabetes mellitus with obesity (lipogenous diabetes); (6) they are most frequent and most intense after the age of fifty.

PHYSICAL SIGNS.

Upon physical examination the most striking features are the general obesity; the relative weakness of the skeletal muscles; the groups of dilated venules, especially the "Bardolphian" "butterfly" area of dilated venules about nose and cheeks, as well as similar areas along the attachment of the diaphragm and elsewhere. According to Hirsch, dilatation of the superficial veins in the subcutaneous fat is a premonitory sign of cardiac weakening; but this is certainly not the case always. Often there is no visible apex impulse; the relative cardiac dulness is increased to both left and right, owing to the transverse position; the cardiac flatness is diminished. The heart sounds usually have a distant character and may be free from murmurs. Occasionally there may be slight œdema of the feet and a small amount of albumin in the urine. In advanced cases of cardiac insufficiency the patient may become much thinner (owing to diminished absorption of fat from the intestine, see page 159), but the pericardial fat may remain undiminished.

TREATMENT.

The treatment of cardiac weakness of fat persons depends entirely upon the stage at which the patient is seen. If œdema and persistent dyspnœa or palpitation upon slight exertion are already present, the case must be treated exactly like one of cardiac overstrain or heart failure from any other cause manifesting similar symptoms, except that, owing to the frequent atrophy and infiltration of the heart muscle, drugs of the digitalis group are often of little use and may even be harmful. The patient should be put upon rest, restricted diet, with liquids restricted to 1000 c.c., purged freely, and bled if symptoms of failure of the right heart set in. Amyl nitrite, nitroglycerin, and erythrol tetranitrate may be used to relieve attacks of dyspnœa, and massage, passive movements, and finally resisted movements, and cold water or Nauheim baths when the patient is able to get out of bed.

When, as is usually the case, the patient is seen before the stage of actual heart failure has set in and is suffering only from what may be considered as the premonitory symptoms of cardiac affection,—palpitation and shortness of breath on exertion, weakness, and giddiness,—the treatment should then be directed toward the obesity rather than toward the heart. A main indication is then gradually to restrict the diet to a heat equivalent of about 1200 to 1700 calories, of which 500 calories (about 120 Gm., 4 oz.) should be proteid (v. Noorden).

(1) **Restricted Diet.**—Numerous restricted diets have been laid down, especially by Banting, Oertel, Hirschfeld, Kisch, and Ebstein. The restriction should not take place suddenly, for fear of weakening the patient, but should take place in several stages, reducing 500 calories each week until the lower limit is reached.[1]

(2) Liquids should be restricted to less than 1000 c.c. (1 quart) per day; this also should be done gradually.[2]

Sample Diet.—V. Noorden gives the following outline diet, which is very satisfactory as a basis capable of modification

		Prot.	Fat.	Carb.	Cal.
8 A.M.	Breakfast—				
	80 Gm. cold lean meat	30.5	1.4		
	½ white roll (25 Gm.)	1.8	0.2	14	205
10 A.M.	1 egg	6.5	6.1		85
12 M.	1 cup lean bouillon		0.8		7
1 P.M.	1 small plate clear soup	0.8	2.0	4	
	150 Gm. (5 oz.) lean meat or fish	57.3	2.8		
	100 Gm. potatoes	1.9		180	
	Peas, beans, cauliflower, asparagus	3.0	10	15	
	100 Gm. fresh fruit	0.5		8	583
3 P.M.	Black coffee		....	....	
4 P.M.	200 Gm. fresh fruit	6		16	90
6 P.M.	250 c.c. (1 glass) skim-milk	6.8	2.0	12	97
8 P.M.	Supper—				
	125 Gm. cold lean meat with pickles	36	3		
	Red beets, radishes, etc.	2		5	
	30 Gm. graham bread	2.0	0.3	12	
	2–3 teaspoonfuls boiled fruit (no sugar)	0.5	....	8	209
	Total	155.6	28.6	112	1087

[1] 100 Gm. (3 oz.) raw meat (proteid 20 per cent., fat 1.7 per cent.) = 100 cal. 100 Gm. (3 oz.) cooked lean meat (proteid 37 per cent., fat 2.5 per cent.) = 175 cal. (about 25 per cent. higher in well-done roasted meats). 100 Gm. (3 oz.) cooked meat of stall-fed animals (no visible fat) (proteid 36 per cent., fat 6 per cent.) = 200 cal. 1 egg (6.5 Gm. proteid + 6.2 Gm. fat) = 85 cal. Cheese (proteid 28 per cent., fat 30 per cent., carbohydrate 2 per cent.) = 400 cal. Milk (proteid 3.4 per cent., fat 3.0 per cent., sugar 4.5 per cent.) = 60 cal. per 100 c.c. 20 cal. per ounce). Potatoes 100 Gm. (3 oz.) = 80 cal. Bread (proteid 7.9 per cent., carbohydrate 35 to 80 per cent., the latter in zwieback and dry breads, 100 Gm. = 200–350 cal. Sugar 100 Gm. = 400 cal. Butter 100 Gm. = 930 cal.

[2] Oertel and Schweninger thought that drinking water is a factor producing fat. Straub and others have shown that this is by no means the case. The only influence of the water lies in the fact that when a meal is taken dry the appetite is less than when water is taken, and consequently less is eaten. However, considerable amounts of fluid increase the volume of blood and the work of the heart, and hence the limitation of fluid saves the heart in this way.

(3) **Increased Exercise.**—Increase exercise gradually as much as possible, especially by walking, either on the level or on gentle gradual ascents, interrupted by frequent rests before either weariness or shortness of breath sets in.[1] In this way the energy used by the body, and hence also the fat burned up, can be materially increased.

	Walking on Level.			
	Per mile.		Per hour.	
Weight of patient	150 lbs.	200 lbs.	150 lbs.	200 lbs.
Rate 2.7 miles per hour	60 cal.	85 cal	170 cal (18 Gm. fat)	225 cal. (25 Gm. fat)
Rate 3.4 miles per hour...	75 cal	100 cal	230 cal (30 Gm fat)	310 cal (40 Gm. fat)

In walking up grade the energy used up is equal to elevation × weight of patient plus the energy expended in traversing the distance; but this is theoretically equalled by the energy saved in the subsequent descent, and on the other hand, both are increased by bringing into play a different group of muscles, these factors can scarcely even be approximately estimated in the individual case. However, Zuntz gives the following empirical figures: a man, 150 lbs., climbing 3 kilometres (1.8 miles) in one hour upon a 10 per cent. grade uses up about 28 Gm. (almost 1 ounce) of fat.

(4) Resisted movements (Schott) carried out under the supervision of an attendant; or contraction of antagonistic muscles (Herz) (see page 194)

(5) Nauheim baths (see page 201) or daily cold baths as cold as can be borne by the patient without shock

(6) **Drug Treatment.**—Strychnine may be administered to increase muscular tone, provided this does not also increase the appetite too much.

Thyroid extract and other "antifat" medication should be scrupulously avoided. Metabolism experiments have shown that the administration of thyroid substance, though increasing the oxidative processes causes a splitting of proteid to a greater degree than of fat, and hence defeats its own end, namely that of burning up the fat without affecting the muscle. It also brings about palpitation, tachycardia and other distressing symptoms and tends to increase rather than to diminish the cardiac features, even though it may be diminishing the obesity itself.

In the obesity of the menopause, tablets of ovarian extract are used to increase oxidation, as this effect has been demonstrated in animals, but clinically the results from its use are rather uncertain

FATTY DEGENERATION

PATHOLOGY.

Pathological Anatomy.—In the condition known as "fatty degeneration" the fat is deposited not by an increase of adipose tissue but in the form of fine droplets within the heart muscle-cells (Figs. 141 and 142).

In some cases these droplets can be seen to almost fill the entire cell, in others they appear as a few diffusely scattered droplets in the sarcoplasm

[1] It must be borne in mind that sclerosis of the coronary arteries is a frequent concomitant of heart weakness in fat people, and hence sudden overexertion or severe exercises are to be avoided at least until the physician has thoroughly acquainted himself with the patient's condition and endurance

Not all the cells are invaded by the fat, but with the naked eye yellow areas of fatty degeneration may be seen mingled with normal areas of red-brown color, which appear normal in structure under the microscope.

As regards distribution, Ribbert recognizes three types (1) diffuse general fatty degeneration, in which all the cells are loaded with fat; (2) mottled degeneration, occurring in the areas which lie midway between or at points most distant from the larger arteries; occurring especially in anæmic individuals and in persons whose blood-pressure is very low, so that the cells which are most distant from the arteries suffer from ischæmia; (3) mottled perrarterial fatty degeneration produced by the action of poisonous substances in the circulating blood, such as phosphorus, arsenic, bacterial poisons, etc., in which those cells suffer most which are brought most closely into contact with the poison, i.e., the cells lying in the vicinity of the larger arteries, while the areas remote from these vessels are normal or involved to a lesser degree.

Nature of Fatty Degeneration. — The fatty degeneration may go on in hearts otherwise healthy in connection with infectious diseases, or in chronic myocarditis and in valvular heart diseases. The exact nature of the process is not clear. Virchow termed it a "degeneration," but this term, although in very general use, does not seem to designate accurately the process. It appears to be a disturbance of cellular metabolism rather than a degeneration of cell protoplasm, and it has been suggested that perhaps this is due to some interference with the oxidizing enzymes such that the fat cannot be oxidized, just as the sugar fails to be oxidized in diabetes. But this suggestion is not founded upon any experimental data.

It is therefore most important from the stand-points of both pathology and prognosis to learn where this fat comes from and how it is formed. Virchow was the first to teach that there was a true fatty degeneration, that is that the fat was formed from non-fatty (probably proteid) substances of the sarcoplasm. It must be borne in mind that the fat might be present in combination as it is in lecithin without being visible, but that it may become visible when it is split off from the lecithin molecules and deposited as highly refractive droplets of true fat.

However, the analyses of numerous observers (Bötteher, Krehl, Rosenfeld) show a definite increase in the fat present in the heart muscle in fatty degeneration. Indeed, according to Rosenfeld, the muscle shows "fatty degeneration" whenever it contains more than 15–17 per cent. of fat within the muscle-cells (in marked fatty degeneration usually 20–21 per cent.). As he put it, "there is no true fatty degeneration, but the cell becomes poor in proteid and fat enters it." That this fat is not derived from the breaking down of cell substance, but is derived either from the fat of the food or from that transferred from the subcutaneous tissue elsewhere in the body, has been shown in many ways. In the first place, Krehl demonstrated that the lecithin content of the heart muscle was practically constant and quite independent of the degree of fatty degeneration and hence that the fat was not derived from this source. Secondly, Rosenfeld showed that in a heart whose left ventricle appeared normal, but whose right ventricle was very yellow in appearance (and showed fatty degeneration on section), the nature of the fat was identical in both. Thirdly, it was shown also by Rosenfeld that if dogs were starved until their subcutaneous fat had disappeared and were then poisoned with phosphorus, the fatty degeneration did not then appear as it did in well-fed dogs. This fact was further demonstrated by Leick and Winckler, who poisoned their dogs with phosphorus and then fed them on mutton tallow (iodine absorption coefficient 38.2), and obtained a deposit within the heart muscle not of dog fat (I. A. C. 58.6) but of mutton tallow. This seems to prove that the "fatty degeneration" of heart muscle is simply a deposit of fat within the muscle-cell just as it occurs within the connective-tissue cell under normal circumstances. The deposition of this fat is not associated with any

change in the lipase of the heart muscle nor of the liver in spite of the apparent increase in fat metabolism. The author also found that the amount of lipase in the lean areas of a human liver mottled with fatty degeneration was the same as in the neighboring yellow areas. It would appear therefore, that, enzymically, the primary change being absent, fatty degeneration lies not in the heart but elsewhere in the body. This is further borne out by the fact that in animals poisoned with phosphorus, oil of pulegon, etc. the total amount of fat in the body is diminished while that in the heart and liver is increased. The latter organs seem merely to deposit the fat thrown into the general circulation.

ETIOLOGY.

Fatty degeneration in the human heart occurs most commonly in association with alcoholism, either acute or chronic, primary and secondary anemias, after hemorrhages, in association with myocarditis, valvular and other cardiac lesions, in most infectious diseases, in miners, smelters, and many metal workers, as well as in numerous other industries where poisonous substances are employed. In a number of cases of death from chloroform anaesthesia fatty degeneration has been found and is usually ascribed to the action of the chloroform, but Rosenfeld believes that in these cases the fatty degeneration is always present before the chloroform was given, and that this fact accounts for the death of the patient.

Not infrequently, as in cases of phosphorus poisoning and of infectious diseases, the same agent which brings about the fatty degeneration also gives rise to diminished tone of the vasomotor centre. Failure of the circulation may result from the latter factor, but this need scarcely be ascribed to the fatty change in the heart.

STRENGTH OF HEART WITH FATTY DEGENERATION.

These results of chemical investigation also find their parallel in the effects upon muscle. Welch in 1888, was able to show that the hearts of rabbits rendered fatty by prolonged exposure to high temperatures were quite normal as regards preservation of blood-pressure, reactions to vagus stimulation, etc., while Hasenfeld and Fenyvessy ten years later showed that animals poisoned with phosphorus withstood the strain from clamping the abdominal aorta quite as well as did normal animals. On the other hand, de la Camp compelled his phosphorus dogs to run a tread-mill until fatigue set in, and found with the X-ray that their hearts had dilated, whereas those of normal dogs did not dilate under these circumstances. The tonicity of the cardiac muscle was diminished. De la Camp's experiments have not been repeated as yet, but they seem to have been very carefully carried out. It seems certain that, as Kraus claims, there is a considerable difference between the endurance of normal hearts and of those with fatty degeneration.

Moreover, patients with fatty degeneration of the heart are very sensitive to digitalis and are frequently injured by it. Sudden death from overdose of digitalis or from acute cardiac overstrain is more common in patients with fatty degeneration of the heart than in almost any other condition. The relative frequency with which fatty degeneration is associated with spontaneous rupture of the heart is also evidence of weakness of the walls.

SYMPTOMS AND SIGNS.

The most characteristic symptoms associated with the condition are those of general debility and feebleness, more or less languor and somnolence, as a rule without marked cardiorespiratory symptoms except shortness of breath on exertion. The pulse is usually small, rather collapsing, and feeble; the blood-pressure is below normal, except when complicated by chronic myocarditis or valvular lesion (maximal pressure 90 to 115 mm. Hg); the pulse-rate is increased. On physical examination the heart may be either normal or dilated, the sounds either feeble and distant or short and sharp; the apex impulse may or may not be well marked. The liver and spleen are often enlarged as part of the general malady of which the cardiac condition also forms a part. There is sometimes œdema of the feet and ankles. However, it must be frankly admitted that none of these is either constant or characteristic; and the diagnosis may have to be made from inference only.

DIAGNOSIS.

The diagnosis of fatty degeneration may often be made with more or less probability from a knowledge of the etiological factors but not from any of the physical signs, so that, as Krehl puts it, **there are no clinical signs for the diagnosis of fatty degeneration of the heart.**

TREATMENT.

When the condition is recognized, or rather suspected, the treatment consists of absolute rest in bed for at least two weeks after the acute disturbance has passed off and until slowed respiration and increased tolerance to mild but gradually increasing arm exercises show that the heart muscle has regained its normal condition. Whether it is possible to overcome the fatty degeneration of a chronically diseased heart is questionable, but in that, as in other conditions, treatment must be guided by the general response of the patient, and over-exertion must constantly be shunned.

It must be borne in mind that hearts which are in a state of fatty degeneration are particularly sensitive to digitalis; so that, when this condition is suspected, digitalis should be either avoided or given in smaller doses than usual.

PROGNOSIS.

Spontaneous recovery is the rule if too great a burden is not imposed on the heart, but in spite of the results of animal experiments, especially those of Welch and Hasenfeld and Fenyvessy, attention must be called to the fact that sudden death is far from a rare occurrence in hearts with fatty degeneration. It occurs most frequently after or during exertion. One can scarcely avoid the suspicion that perhaps the condition which brings about the change in the fat metabolism is also one which limits the total metabolism of the heart muscle-cells and consequently their contractility; so that after a certain limit is passed they suddenly cease their function, just as is the case in the cellular asphyxia of intermittent claudica-

tion and coronary sclerosis (see page 282, Fig. 166), or in toxic myocarditis from diphtheria. Spontaneous rupture of the heart is particularly common in cases of fatty degeneration. The latter was present in 77 per cent. of the cases collected by Hamilton.

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IX.

AFFECTIONS OF THE MYOCARDIUM.

A certain amount of degeneration in the fibres of the heart muscle occurs during the course of every acute febrile disease or intoxication. As has been seen in previous chapters, the heart under these conditions exhibits signs of overstrain, and the diagnosis of myocarditis, therefore, depends upon the degree rather than the mere existence of cardiac weakness. However, in certain cases the signs of cardiac weakness overshadow those of the original disease and it is in these that acute myocarditis is usually recognized. The chronic changes, however, which follow long after the original disease has subsided, present a less complicated picture and therefore are more easily recognized.

PATHOLOGICAL ANATOMY.

The lesions of acute and chronic myocarditis are merely different stages in a process which is more or less continuous. The lesions of chronic myocarditis are always preceded by the acute lesions, but the degeneration may not be so severe at any of the earlier periods as to give rise to symptoms of cardiac weakness.

In the first stage of acute myocarditis there is injury and degeneration of the muscle-fibres, with œdema about them, and infiltration of polymorphonuclear or mononuclear cells into the œdematous spaces between the fibres.

Degenerative Changes. The degenerative changes which take place in the heart muscle are, (1) parenchymatous degeneration, (2) fatty degeneration, (3) hyaline and amyloid degeneration, (4) calcareous degeneration, and (5) fragmentation.

Parenchymatous degeneration of the heart muscle was first described by Virchow and Boettcher. The muscle-fibres swell, lose their striation, and the plasma contains numerous granules of an albuminous material, probably altered muscle proteid (myosin). They retain their contractile power to a certain extent, but its force at this stage is somewhat impaired, and the cell may subsequently return to normal without undergoing complete necrosis. In the more severely injured cells the nucleus is destroyed, the sarcoplasm becomes filled with vacuoles, takes on a basic stain, and is gradually absorbed, leaving only the sarcolemma. Often, but not always, parenchymatous and fatty degeneration go on in the same fibre, the fat being deposited as the proteid is removed.

In some cases fibres undergo hyaline or waxy degeneration (Zenker and present an absolutely homogeneous appearance, taking up the acid stains (protoplasmic) with great avidity. In rarer cases there is a calcareous degeneration with deposit of calcium salts in the muscle-cells. These cells then take up the basic (or nuclear) stains (description of

a case and discussion of the literature is to be found in the article of E. K Cullen). The degeneration is never uniformly distributed throughout the cells, not all the cells being affected at once or in the same degree.

Occasionally the heart muscle-cells show peculiar splits extending transversely across the whole or part of the cell This condition is known as "Fragmentation" The fibres may show no other signs of degeneration, the transverse striations may be clear, and the longitudinal striæ may be distinct up to the line of the fracture Fragmentation has been found after death from a tremendous variety of causes, even in individuals dying from accident It does not seem, therefore to be a sign of specific degeneration Dietrich's attempts to prove it an artefact and either to exclude it when once present or to bring on fragmentation by allowing the heart to pass through various stages of decomposition have been unsuccessful, and Buhlig in a very careful research seems to have shown that it is an artefact which is produced when the microtome knife cuts at right angles to the muscle-fibres This observation still requires confirmation Otherwise the consensus of opinion seems to be that fragmentation is the result of some change in the muscle-fibres occurring during the death agony, and that it is not to be regarded as a degeneration

As illustrating the frequency of the several types of myocardial degeneration Romberg finds the following frequency in 29 cases

Typhoid fever—11 *cases* Parenchymatous (albuminous) degeneration, moderate or intense 10 fatty, present 6, absent 5; hyaline or waxy (slight 2 absent 9

Scarlet fever—10 *cases* Albuminous degeneration, present 8 absent 1, not noted 1; fatty intense 1, moderate 1, absent 8 hyaline or waxy moderate 3 absent 7

Diphtheria 8 *cases* Albuminous, intense 1, moderate 4, absent 3 fatty, intense 5, absent 3, hyaline or waxy, present 2, absent 6.

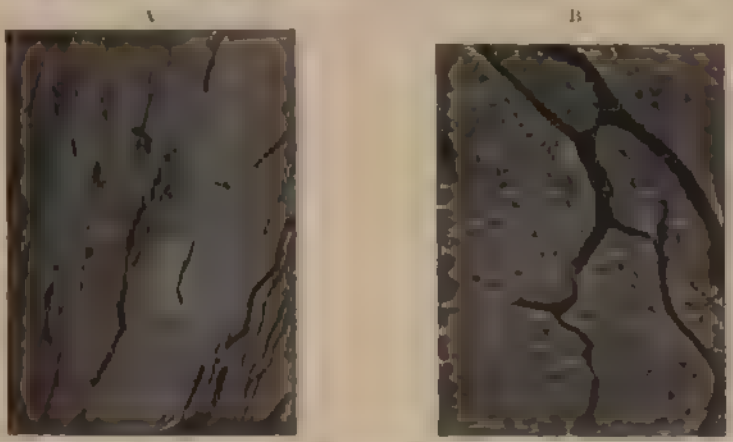


FIG. 144.—Infiltration along the course of the blood-vessels in subacute myocarditis. B, blood-vessels injected. A, Low power. B, same, higher power. Photomicrographs by Dr. P. S. Bond.

Distribution of Myocardial Changes.—In man, according to Krehl,[1] acute myocardial lesions are particularly common in the papillary muscles of the left ventricle and in the mus-

[1] *Examination of Specimens.*—In cases in which the state of the myocardium is of importance the microscopic structure of the heart muscle should always be examined by the method of Krehl. Krehl cuts the heart into cubical blocks 1 cm. in size, numbering them in order so that the exact location of each block can be accurately determined. These blocks are fixed in Müller's solution and a section or two from each is examined. In this way a very thorough study of the extended distribution of lesions may be gained and a study of a very few hearts thus reveals many accurate knowledge that can otherwise be gained from a large number of organs examined less thoroughly

culature about the left auriculoventricular ring. Pearce and Fleisher and Loeb have produced in rabbits myocardial lesions having the same distribution by the injection of adrenalin (Pearce) or adrenalin with spartein or caffeine (Fleisher and Loeb). Roy and Adami have shown that œdema occurs most readily in these regions during experimental cardiac overstrain.

In contrast to the lesions upon the valves, the toxic or infective agents giving rise to lesions of the myocardium do not spread through the walls from the cavity of the ventricles, but are carried into the heart muscle through the coronary arteries and distributed through their finer branches. The foci of inflammation whether of bacterial or toxic origin usually arise within the lymph spaces around the arteries, which they surround in sleeve, cuff, or signet-ring distribution.

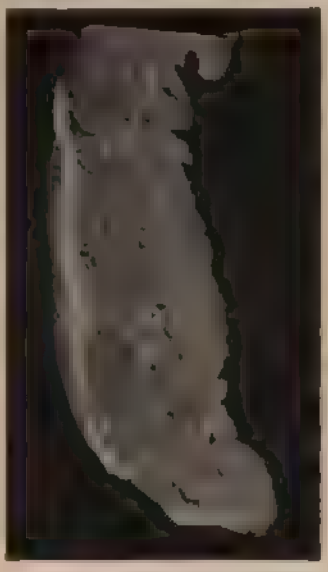


Fig. 145.—Septic myocarditis with multiple abscesses in the heart wall. The arrows point to the abscesses.

Abscess. The form which the foci assume depends chiefly upon the nature and properties of the infective agent. If the virulence of the germ is great, abscesses may be produced in the heart muscle (suppurative myocarditis) as elsewhere in the body. These abscesses are usually produced by small septic thrombi which plug the minute branches of the arteries. Under the influence of the fibrin ferment secreted by the bacteria, the vessel soon becomes completely filled with a thrombus, an area of ischæmia results in the heart muscle, which quickly becomes infected and breaks down to form an abscess. These abscesses vary in size from a submiliary nodule to a cavity separating the muscle layers in the entire interventricular septum. They are usually produced by the pyogenic cocci in septicæmia or following trauma to the heart (see page 519). The outcome is usually fatal. Occasionally there is rupture of the ventricle through the necrotic portions of the wall.

"Rheumatic" Foci. In the less virulent infections, such as rheumatism, typhoid fever, influenza, the foci do not undergo suppuration, but the lymph spaces around the arteries and capillaries are filled with cellular infiltration, polymorphonuclear in most of the acute infectious diseases, while mononuclear cells predominate in myocarditis from typhoid fever and subacute rheumatism.

Since rheumatic fever is perhaps the most common cause of myocarditis, the lesions which it produces are of particular interest. Romberg, Aschoff, Geipel, and Coombs have called attention to the presence of small submiliary foci 0.1–0.2 mm. in diameter, which occur with great frequency in rheumatic patients, especially in the musculature about the mitral ring. Each focus consists of a hyaline centre formed by agglutinative thrombosis within a capillary. About this there is a zone of giant cells each containing

2–4 nuclei, and these in turn are surrounded by a wider zone of mononuclear cells interspersed with eosinophiles. The writers mentioned regard these foci as pathognomonic of rheumatism, although they may bear only the general features of a subacute inflammation about an area of hyaline thrombosis. Indeed the most typical specimen of these found in the Johns Hopkins Pathological Museum was seen in a case of non-rheumatic myocarditis. On the other hand, Freund has reported a case of acute rheumatic myocarditis in which the infiltration was mainly polymorphonuclear.

Bracht and Wachter have recently produced arthritis, endocarditis and myocarditis with lymphocytic infiltrations in animals by injection of cultures of diplococci obtained from two cases of acute articular rheumatism. These infiltrations contrast sharply with the polymorphonuclear infiltrations usually produced by pyogenic streptococci.

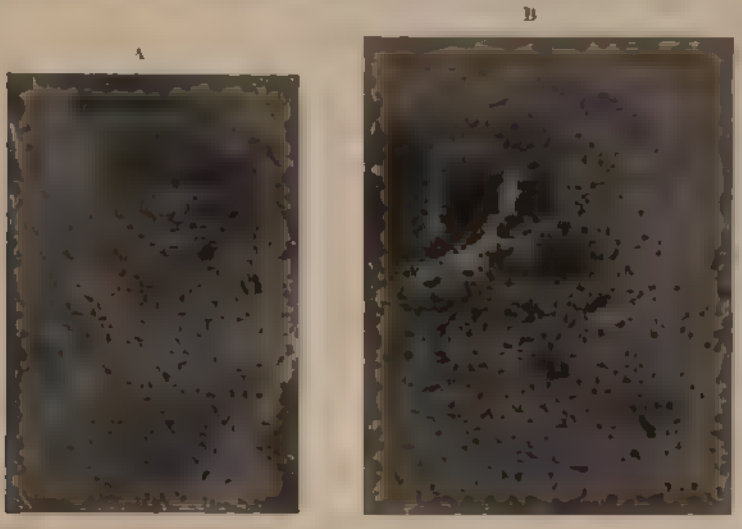


FIG. 146.—Photomicrograph, showing an abscess in the heart muscle. A Low power. B Same higher power.

Subsidence of Lesions.—The changes which occur in the myocardium when the patient recovers from the acute infection or intoxication, which is the causal factor, vary both with duration and intensity of the disease and the rapidity and completeness of the recovery. If the causal factor completely disappears and its sojourn in the body has been a short one, no permanent changes may have taken place. The œdema of the fibres disappears, the cellular exudate may be absorbed *in toto*, and the myocardium may resume its normal appearance. If areas of fibres have been destroyed their place may be taken by scar tissue. But if the duration of the process has been so long that connective tissue has begun to be formed in the exudate, the traces are no longer obliterated and a chronic myocarditis has set in.

PATHOLOGICAL PHYSIOLOGY.

As has been seen in previous chapters, hearts whose muscle is injured become dilated upon comparatively slight exertion, while healthy hearts resist dilatation in spite of tremendous

exertion. Moritz and Dietlen, whose X-ray studies have demonstrated that the normal heart becomes smaller in severe exercise, have shown that, on the contrary, the heart whose muscle is diseased undergoes tremendous dilatation. Nevertheless it may maintain a normal or even heightened blood-pressure without apparent effort, and except for the dilatation may present no other signs of abnormality. It is more common in conditions of acute myocardial change to have a low blood-pressure, but this is due to the fact that the toxic substances which injure the heart muscle also depress the vasomotor centre. The low blood-pressure is due to the latter influence and not to the weakness of the heart.

These facts were brought out by very interesting studies of the physiology of the heart muscle after injections of diphtheria toxin which were made by Rolly and later by v. Stejskal. Rolly used a dose of toxin which just killed his rabbits in twenty-four hours, and then began his experiments about twenty-two hours after the injection. He found that at this time the blood-pressure and pulse-rate of the animal were still quite normal, and that the heart was still able to respond well to increased work thrown upon it by compressing the abdominal aorta, etc., and that the blood-pressure increased considerably. About half an hour before death, however, the blood-pressure began to fall, owing to loss of vasomotor tone, as had been shown by Romberg. Even at this time the heart was still strong enough to respond by a second rise of blood-pressure upon clamping the abdominal aorta. Very soon after this, however, within a few minutes, the rate became irregular and the heart weakened completely. V. Stejskal's results were similar. The action of the diphtheria toxin had not been immediate, but it had required several hours to combine with the heart muscle, after which its weakness was manifest.

The conclusion reached by Rolly and v. Stejskal is that the heart remains competent in spite of muscular weakness until a certain degree of strain is imposed upon it, when it suddenly crosses the threshold that leads to failure, dilatation, and even death. The threshold of cardiac overstrain in the healthy heart is at a much higher level.

Arrhythmia in Acute Myocarditis.—Irregularity of the pulse cannot be brought about by injuring the myocardium by injection of alcohol, iodine, or even KCN, but often occurs in man as a result of myocardial lesions, especially after exercise and overstrain. Gerhardt, Müller, and Schönberg have called attention to the association of irregularity with structural changes and paralyses of the right auricle. In mitral disease it is probable that irregularity arises in the left auricle rather than in the right, since the latter is then not the seat of pathological conditions.[1]

Bradycardia is met with in the late forms of diphtheric, influenzal, and pneumonic myocarditis and occasionally during the febrile stage. It is often vagal in origin, but is sometimes due to depressed conductivity of the auriculoventricular bundle, the ventricle responding only to alternate contractions of the auricle (2 : 1 rhythm). It is probable that under these conditions toxic myocardial changes have taken place in the bundle (Mackenzie). It is not unlikely that some of the sudden deaths during convalescence from diphtheria may be due to this cause (Dunn, see page 478).

[1] More fully discussed in chapter on Mitral Stenosis.

SIGNS AND SYMPTOMS.

The most characteristic sign of myocardial weakness is dilatation of the heart (see page 227). The heart is usually, but by no means always, rapid, the sounds may be clear but are usually short and sharp; they may be embryocardiac in rapid hearts; a gallop rhythm, especially of the presystolic type, may be present, or the sounds may be definitely split (reduplicated). It is also very common to hear soft systolic murmurs over the apex or the tricuspid area, due to functional insufficiencies at the auriculoventricular orifices (see page 140), or to hear the "accidental" systolic murmur in the pulmonary area. The second pulmonic sound is usually accentuated from stasis in the pulmonary vessels.

Clinically, uncomplicated myocarditis is met with in the course of the febrile diseases and the intoxications, especially alcoholism, phosphorus poisoning, and ptomaine poisoning. It is present also in a certain degree in almost every case of acute endocarditis or pericarditis, where it is but part of the general "carditis."

Its manifestations are simply those of acute heart failure or of cardiac overstrain occurring while at rest or upon very slight exertion. The symptoms are, therefore, sometimes those of broken pulmonary compensation (failure of the left ventricle, page 139), sometimes those of broken systemic compensation (failure of the right ventricle), according as the left ventricle or the right is the one most affected. In many cases there are attacks of precordial pain amounting almost to angina pectoris, coming on when the heart is acutely dilated after excitement or exertion.

ACUTE MYOCARDITIS IN RHEUMATIC FEVER.

Although weakening of the heart is one of the most important factors in general asthenia that accompanies or follows tonsillitis or rheumatic fever, it does not often kill the patient and hence is not often a striking feature at the autopsy table.

The following history illustrates the course in fatal cases, showing (1) the gradual insidious onset, (2) shortness of breath, extreme weakness, and finally ascending œdema, (3) dilatation of the heart, with œdema and degenerative changes in the heart muscle, without either hypertrophy, fibrous changes, or valvular lesion.

Case of Acute Rheumatic Myocarditis.

Annie Jones, female, colored, 48, admitted July 5, 1904, complaining of "rheumatism," of which she has had attacks for many years, especially marked during the last two years. The knees and shoulders have been the joints most frequently affected. She has had no other infectious diseases and the previous history is otherwise negative. No shortness of breath nor palpitation. During past four weeks has been compelled to sleep upright in a Morris chair, and has had incontinence of fæces.

Physical Examination.—Patient is a very stout colored woman, lying quietly on her back in bed. Pupils equal and react to light and accommodation. Chest clear.

Heart.—Impulse is not visible. Relative cardiac dulness extends 13 cm. to left of midline in fourth interspace, 30 cm. to the right. First sound at apex

is very loud and not perfectly clear, though there is no definite murmur. Second sound resembles the first in quality but is clear. Pulse regular, of good volume, rather high tension, 100 per minute. Vessel wall somewhat thickened.

Abdomen is extremely large and swollen, there is dulness in dependent portion. Liver is not enlarged. Legs are extremely swollen and indurated; do not even pit on pressure. Knee- and ankle-joints much swollen and stiff. A round perforating ulcer is present at left heel. No disturbance of sensation anywhere.

Temperature 99°; red blood-corpuscles 4,046,000, hæmoglobin 55 per cent.; leucocytes 3800.

Ordered rest in bed; soft diet; diuretin 1 Gm. (gr. xv) q. 4 h.; ulcer of foot to be irrigated with sol. potass. permang. 1:20000 b. d. On July 7, ordered tinctura digitalis 1 c.c. (♏xv) q. 4 h., ad dos. viii; this was then repeated and continued throughout the course of disease. Spts. glycerylis nitrat. gtt. ii, q. 4 h., alternating with sod. nitrit. 0.3 Gm. (gr. v) q. 4 h.; morphin. sulph. 0.008 Gm. (⅛ gr.) p. r. n.

July 12. Heart's action irregular; first sound reduplicated over tricuspid area; no murmurs. July 15. There is a large perforating ulcer just below coccyx. This was irrigated with potass. permanganate 1:20000 and packed with iodoform gauze. July 16. Temperature 106°, percussion note impaired at left base behind where breath sounds are absent. A few râles have previously been heard in this area. Ordered strychnine sulph. 0.003 Gm. (1/20 gr.) and digitalin 0.003 Gm. (1/20 gr.) hypo. q. 4 h. At 7.30 P.M., respiration shallow with expiratory grunt. At 11.00 became unconscious and died at 12.45.

Autopsy showed about 1 litre of fluid in peritoneal cavity; congestion of lower lobe of lungs.

Heart. Several opaque white patches over epicardium, one with a diameter of 3 cm. Coronary arteries soft and smooth. Heart muscle soft, flabby, and of yellowish-brown color, studded with numerous small opaque white areas. The muscle bundles are widely separated nuclei. Under the microscope the muscle-fibres are seen to be swollen, little new growth of interstitial connective tissue. Heart weighs 250 Gm. Slight sclerosis about base of aorta, none elsewhere. Kidneys normal in size, pale and cloudy. Liver shows some fatty degeneration.

Fig. 147.—Orthodiagraphic outlines of the heart of a child during the course of a severe diphtheria. (After Dietlen, *München. med. Wchnschr.*, 1905, in + + + + +, outline on fifth day, MR. = 3.0 cm., ML. = 6.0 cm., L. = 9.1 cm.; — — outline on seventh day (MR. = 3.5 cm., ML. = 8.1 cm., L. = 12.4 cm.); — — outline on twenty-sixth day (MR. = 2.0 cm., ML. = 6.5 cm., L. = 9.3 cm.).

DIPHTHERIC AND INFLUENZAL MYOCARDITIS.

Acute myocarditis is the chief cause of death in diphtheria and influenza. In these conditions it may manifest itself either, (1) as an early form during the course of the fever, or (2) as a late form which becomes manifest after the temperature has fallen. The cases of diphtheric myocarditis have been most carefully studied by Hibbard in 800 cases with 119 deaths (15 per cent.) at the Boston City Hospital. In spite of the high average mortality, the mortality was less than 5 per cent. in those cases in which the pulse-rate was below 130 per minute, increasing as the pulse-rate increased above that figure. Death was especially frequent in those cases in which a gallop rhythm was noted. Bradycardia (under 60 per minute) was not a severe sign in adults (14 cases without a death; only 2 with cardiac symptoms), whereas in cases under 7 years it was a very grave sign (6 cases, 5 deaths). In all Hibbard's fatal cases there were both acute myocardial change and degeneration of the fibres of the vagus.

Sudden death is not uncommon in cases of diphtheric myocarditis, in Dunn's case, from the onset, heart-block (Adams-Stokes syndrome)

was the result of myocardial change in the vicinity of the auriculoventricular bundle. The slow pulse also is often due to partial heart-block, 2 : 1 rhythm, though this may be due to overstimulation of the vagus as well as to injury of the bundle.

Just as diphtheria affects the myocardium in the very young, influenza affects it in the aged. Indeed myocarditis constitutes one of the gravest effects of this disease, and is especially to be feared after the sixth decade.

The following case serves as an example:

Case of Influenzal Myocarditis

Patient, aged 75, of sedentary habits, rather stout, but free from all cardiac symptoms. Pulse had always been of good volume and regular. Had a severe attack of influenza in March, 1903, confining her to bed for a month. No special cardiac features. After a short convalescence she was again able to be up and about. A few days later, just after retiring, she had a severe attack of cardiac asthma, breathlessness, orthopnœa, and slight precordial pain. No true angina. Moderate degree of cyanosis. Pulse small, rapid, irregular. Cardiac dulness slightly enlarged. Soft systolic murmur heard over the entire heart. The attack lasted half an hour, symptoms being much relieved by inhalations of amyl nitrite.

Patient was given complete rest in bed for a few days, with fluidextract of digitalis ♏v (0.3 c c.) three times a day and soft diet, and was then kept at rest in a large arm-chair. Gradual convalescence. Soon became free from symptoms, but pulse remained 70 and irregular and she was compelled to refrain from every effort except one daily trip up and down stairs, during which she rested at each step long enough to count twenty. In June and July, 1904, she had several similar attacks, and though she improved somewhat her pulse remained permanently irregular. Died suddenly a year and a half later, death following six weeks after a severe cellulitis of the leg.

Case of Subacute Alcoholic Myocarditis

B. C. S., reporter, married, aged 36, admitted to the service of Prof. J. O. Hirschfelder, City and County Hospital of San Francisco, January 23, 1905, complaining of shortness of breath and swelling of feet. Father and brother are subject to rheumatism, and patient himself had swelling of joints four years ago, about the time of a gonorrhœal infection. He had measles, whooping-cough, and scarlet fever as a child, and typhoid fever seven years ago. Denies syphilis. Married, but has had no children. Uses tobacco in moderation, but drinks whiskey in excess, as a probable result of which he has fallen from the best to the lowest strata of society.

Present Illness.—Four weeks ago while in the midst of a series of debauches he noticed that his shoes became tight, and in a few days his legs became so swollen that he could not put on his drawers. He had pain in the legs on walking, owing to the œdema. He also felt very weak and became exhausted easily. Has had shortness of breath on exertion.

Physical Examination.—Well-nourished man of good color. Tongue and uvula deviate slightly to the right. General glandular enlargement. Epitrochleans palpable. Chest negative except for a few moist rales over right axilla and base.

Heart.—Cardiac impulse not visible. Relative cardiac dulness extends to 1.2.5 cm. from mid-line in fifth interspace 13 cm. outside mammillary line, 4 cm. to right of midline and above to the third rib. Sounds are very rapid; the first sound everywhere replaced by a systolic murmur which is loudest at the apex, not transmitted to the axilla; pulmonic second accentuated. Pulse 108, regular in force and rhythm, low tension, fairly good volume. Radial artery not palpable.

Liver just palpable. No scar on genitalia. Lower extremities are covered with pediculi and raw scratch marks. Marked œdema of both legs. Urine negative, sp. gr. 1028.

Ordered tinct. digitalis 0.6 c.c. (♏x) q. 4 h.; spir. glyceryls nitratis 1 gtt. q. 4 h.; sol. magnes. sulphat. sat. 30 c.c. (℥i), ung. zinci oxid. to legs.

Jan. 30. Pulse slow and somewhat irregular, venous tracing showing that some of the auricular impulses did not reach the ventricle (2 : 1 heart-block). Given atropine 0.0015 Gm. (1/40 gr.) at 12.45 P.M. At 2.00 P.M., max. pr. 135, min. 75/80. Pulse-pressure 60; pulse-rate 60–5000. Pulse-rate absolutely regular, as shown at the brachial artery tracing taken at 1.45 P.M. Digitalis was now discontinued.

Feb. 2. Oedema gone. Soft systolic murmur still present at apex. Pulse-rate 72, absolutely regular, responding to all impulses from the auricle. It never again became irregular.

Feb. 11. Feels quite strong. Up and about. Heart has been regular and all murmurs gone.

March 2. Has had slight swelling of feet. Was again put to bed. The swelling disappeared within 24 hours.

In a few days the patient was again up and about, and in a week or ten days later was allowed to continue his work in the pantry. Was discharged apparently cured about May 15.

DIAGNOSIS.

As has been seen, the diagnosis of acute myocarditis in many cases is made more by inference than by definite signs. The presence of symptoms of cardiac weakness in an infectious disease, out of proportion to the severity of the latter or to the apparent severity of the endocardial lesion, is presumptive evidence of severe myocardial involvement. The symptom-complex of restlessness or marked dulness, constriction over the chest, and precordial pain, vomiting, cyanosis, and increase in the area of cardiac dulness, during or after an attack of an infectious disease or of delirium tremens, is practically pathognomonic. The presence of a systolic murmur at the apex and over the body of the heart, which may even be transmitted to the axilla but which disappears during convalescence, added to the other symptoms above mentioned, would indicate myocarditis rather than endocarditis. It must be borne in mind, moreover, that the presence of true endocarditis or pericarditis is evidence in favor rather than against the presence of an additional myocarditis, and that in the acute form the symptoms are quite as liable to be due to the insufficiency of the muscle as to the valves. On the other hand, just as a most acute nephritis may be present without the presence of albumin or casts in the urine, so acute myocardial changes may be present without definite signs of cardiac weakness other than a tendency to fatigue. In view of the observations of de la Camp, Moritz, Dietlen, and Hornung, myocardial changes may be diagnosed in cases in which the heart undergoes transitory dilatation (with or without transitory valvular insufficiencies) upon comparatively slight exertion. The cardiac area under such conditions must be most carefully outlined, if possible with the orthodiagraph. In the absence of the latter careful percussion may often suffice. The changes must be 1 cm. or more before they should be considered as definite.

TREATMENT.

The management of a case of acute myocarditis differs essentially from that of the chronic form, owing to the fact that in the former the changes in the muscle may be of a temporary character, while in the latter the changes are permanent. Accordingly, in the acute form the aim is to allow the muscle to return to its normal state,

while in the chronic form this cannot be hoped for, and the treatment is directed toward obtaining the best functional result possible in the changed muscle that is left. The one aims at bringing about subsidence, the other at inducing hypertrophy.

Accordingly, even in the mildest form of acute myocarditis rest is all-important — complete rest in bed until the degenerative changes in the muscle have subsided. This is especially important, since cardiac overstrain sets in very easily in such hearts, and it is probable that this, in even the slightest degree, increases the injury to the muscle-fibres as well as the extent of the interstitial oedema and infiltration. The patient should be kept in bed at least two weeks after any indications of myocardial weakness have subsided, and if possible until the pulse-rate has again become slow. An easily digestible diet equivalent to about 1000–1500 calories should be enforced (see page 167), frequent feeding of small quantities being resorted to in the place of three comparatively large meals.

An ice-bag should frequently be applied to the precordium, since it tends to slow the heart-rate. Some writers, especially Caton, strongly favor the application of small blisters to the precordium and the administration of small doses (0.3 Gm. or 5 gr.) of potassium iodide, but it is extremely doubtful whether this has any effect upon the course of the disease.

If anæmia arises, iron should be ordered in some form, usually as Blaud's pills.- ferri carbonas saccharatus (0.25 Gm., 4 gr.), or Vallet's mass (same as Blaud's pills with honey instead of sugar but more permanent), or elixir ferri, quininæ et strychninæ phosphatum (4 c.c., 1 fluidrachm). If constipation or other digestive disturbances result, hæmatin or some other "organic" iron preparation, that is, where the iron is combined with proteid. The patient's bowels should be kept freely moving without effort, best by means of Rochelle salts, sodium phosphate, Epsom salts, or Seidlitz powders. The effervescent citrate of magnesia usually causes greater abdominal distention than is desirable, owing to the upward displacement of the diaphragm.

Hypersensibility to Digitalis. — The usefulness of digitalis in acute myocarditis is a debatable question. Digitalis acts as a spur to the heart and raises the strength of the contraction until it enables the fibres to draw on their reserve force at each contraction, but it does not raise the limit strength. When that limit is already approached it spurs them too far, and drives them to overstrain and even to death.

Whether, in any individual case, digitalis will do good or harm will depend, therefore, upon the degree to which degenerative changes have progressed and the amount of reserve force that is left. Thus, in the case of B. C. S., the myocardial degeneration was slight and the beneficial action of digitalis was marked. With A. J., however, the case was different. Degeneration had reached too advanced a stage and the drug was useless, perhaps even harmful.

Even the heart of B. C. S., however, manifested the abnormal susceptibility of such hearts to digitalis, since it produced partial block and extrasystoles with doses which barely sufficed to slow the heart of the average patient.

Moreover, in acute myocarditis the heart is hypersensitive to digitalis. For example, in the case of B. C. S., a normal dose produced an abnormally intense reaction with signs of the first stage of digitalis poisoning—partial heart-block and extrasystoles. Fortunately in this case the good effects outweighed the bad, but it belonged to the group of cases which prove conclusively that in acute myocarditis digitalis should always be given in smaller doses than would be used for a heart with a valvular lesion which showed the same degree of heart failure.

Strychnine.—As regards strychnine, both its beneficial and its harmful effects are less marked than those of digitalis. It is therefore less liable to overstep the limit of tolerance. In ordinary doses it tends to increase the cardiac tonicity, as well as to stimulate the cardiac nerves, the respiratory and vasomotor centres, so that it becomes a valuable drug in such conditions.

CHRONIC MYOCARDITIS

PATHOLOGICAL ANATOMY.

Pathologically the chronic inflammatory changes in the myocardium may be divided into three groups:

1. Cicatricial patches or scars arising from the healing of isolated areas of inflammation (abscess or focal infiltrations) or from the organization of areas of infarction.
2. Thickening of the septa that separate the muscle strands (interfascicular myofibrosis Deluo) occurring when the heart muscle hypertrophies.
3. Diffuse degeneration of the muscle-fibres with invasion of the fibre bundles by strands of connective tissue (*cardiosclerosis*, Huchard, *interstitial myofibrosis*, Deluo).

FIG. 148.—Specimen showing a cardiac aneurism covered with pericardial adhesions.

FIG. 149.—Chronic myocarditis (cardiosclerosis).

Cardiac Cicatrices.—The areas of cardiac cicatrices are quite common in coronary sclerosis, in which they represent the site of healed infarcts in the area supplied by the affected artery. The fibrous tissue composing the scar, relatively poor in elastic fibres, is weaker than the rest of the heart wall, presenting the condition termed by Ziegler myomalacia cordis, and it may bulge out to form an aneurism of the heart (Fig. 148).

Spontaneous rupture occurs in such areas, and death occurs from hemorrhage into the pericardium, though, according to Hamilton, this is not as frequent a cause of spontaneous rupture as is fatty degeneration.

On the other hand, the smaller areas of cicatrization may represent complete obliteration

Interfascicular Connective-tissue Proliferation. Interfascicular myofibrosis or hyperplasia of the septa between the bundles is to be regarded as a concomitant of cardiac hypertrophy, and represents a strengthening rather than a weakening of the heart.

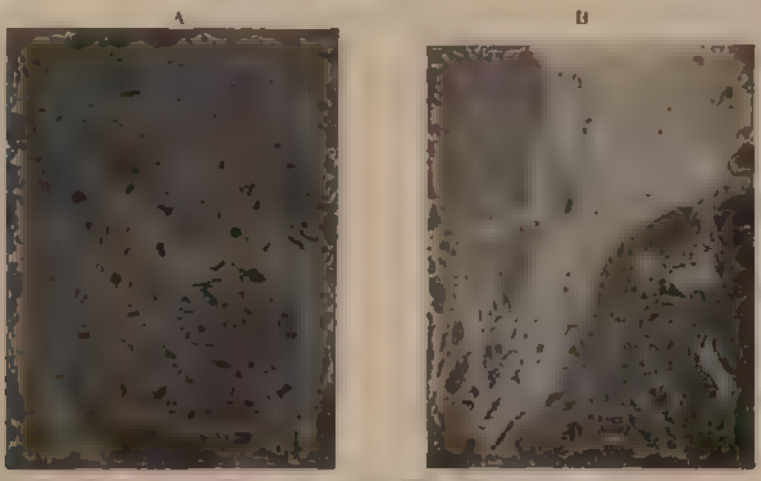


FIG. 170.—Specimens showing chronic myocarditis. Photomicrographs by Dr. Chas. S. Bond.) A, Interfascicular myofibrosis, penetrating into the bundles of muscle-fibres. Hypertrophy of some fibres, atrophy of others. B, Coarse strands of connective tissue penetrating between the bundles of muscle-fibres: interfascicular myofibrosis.

Cardiosclerosis. The most important form of lesion in chronic myocarditis is the interstitial myofibrosis or cardiosclerosis. This form is met with in senile hearts and in most cases of chronic heart failure. According to Dehio, it occurs only in those hearts which have been subjected to long-continued dilatation, frequently in hearts in which hypertrophy has preceded the dilatation. The heart muscle is œdematous. The fibres are found in all stages of change—normal fibres, large healthy hypertrophic fibres, large vacuolated degenerating fibres, and small ones in the various stages of atrophy—in a single microscopic field. Many of them are undergoing fatty degeneration. In response to the well-known biological law that wherever the parenchyma of an organ is gradually destroyed hyperplasia of the interstitial tissue takes its place (Weigert, Dehio) fine strands of connective tissue are seen everywhere winding their way between the muscle-fibres and gradually taking their places.

RELATION OF SITE OF MYOCARDIAL LESION AND DISTURBANCE OF FUNCTION.

Lesions in the Ventricles. Attempts have been made by numerous investigators to demonstrate a definite connection between the exact site of the myocardial lesions and the disturbance of function met with. Krehl, who under Ludwig's inspiration was the pioneer in this field, inau-

gurated the method of studying sections from every part of the heart, and found that the papillary muscles and the musculature about the mitral ring were affected with great frequency; but he was unable to establish more definite relations. Albrecht's attempt to do this for the various muscle layers discovered by Krehl and J. B. MacCallum has called forth a vigorous contradiction from Aschoff and Tawara, who have made a most careful study of 150 pathological hearts by Krehl's method.

FIG. 151.—Hypertrophy of some muscle bundles in the auricle with atrophy (transparency) of other areas. From a specimen in the Army Medical Museum Washington D. C.)

On the other hand, His, Erlanger, Stengel, Schmoll, and a host of others have demonstrated that lesions in the auriculoventricular bundle give rise to heart-block, while Aschoff, Tawara, Saigo, Barker, and Hirschfelder have shown that lesions affecting one branch of this bundle do not affect the contraction of either ventricle. Very recently, however, H. E. Hering has revived interest in these questions by showing upon the excised heart that if the strand of Purkinje fibres (conduction system) to one papillary muscle is cut or injured, that papillary ceases to contract, although the rest of the heart continues to do so.

Lesions in the Auricles.—Studies of lesions in the auricles, though fewer, have been still more remunerative. Dehio and his pupil, Radasewsky, demonstrated that in chronically dilated hearts the myocardial changes in the auricles were much more marked than those in the ventricles; and Schonberg, under D. Gerhardt's direction, has shown that permanent arrhythmia with auricular paralysis is associated with infiltrations of the intervenous area which correspond to the embryonic sinus, the spot at which the cardiac impulse probably originates.

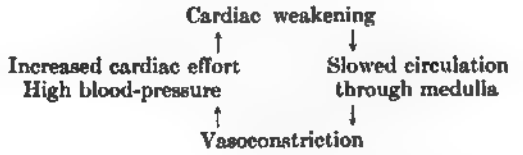
PATHOLOGICAL PHYSIOLOGY.

The chief physiological features of chronic myocarditis are:

(1) Chronic weakness of the heart, with tendency to undergo dilatation and overstrain.
(2) Frequency of extrasystolic or absolute irregularities.

Compensation in Myocarditis. The course and characteristics of myocardial weakness have been fully discussed under the pathological physiology of cardiac overstrain (page 134). Indeed, the persistence of a primary overstrain with the concomitant œdema of the heart muscle may be an important factor in instituting chronic myocardial changes or in rendering the heart especially susceptible to alcohol, toxins, tobacco, or other influences that would otherwise not affect it. The changes in the myocardium, the lowered tonicity, the persistent œdema, the reduction in the number of efficiently contracting muscle-fibres, all tend to lower the threshold of exertion at which overstrain is ushered in. Whether the overstrain manifests itself as a broken pulmonary or a broken systemic compensation, or as both together, depends upon the relative and absolute strength of the two ventricles as well as the nature of the exertion.

Blood-pressure.—The occurrence of such overstrain is, however, quite consistent with the maintenance of a normal or, especially, a high blood-pressure. This high blood-pressure, strange to say, is in itself the result of the chronic cardiac insufficiency and the slowing of the circulation. With the slowing of the circulation there comes asphyxia of the medullary centres, which stimulates them and brings on an intense vasoconstriction. The vasoconstriction narrows the arterial bed so much the arterial pressure must be raised until the blood flows through the medullary centres at the proper rate. The weakened heart must thus rise to the occasion and sacrifice itself to save the medullary centres. The more it fails the more work these inexorable centres demand from it, the more they throttle the arteries in their struggle to get blood from the flagging heart. The more the arteries are throttled the greater the constriction, the smaller the arterial bed, and the less the systolic output necessary to overfill the arteries, the greater the force necessary to drive it. The heart may therefore empty itself incompletely but at high pressure against this high peripheral resistance, while the increase in residual blood within the ventricles leads to dilatation and stasis. This condition of stasis with high pressure, both resulting from chronic cardiac weakness, is usually termed "high pressure stasis" (Hochdruckstauung). Its factors actually constitute a vicious circle:



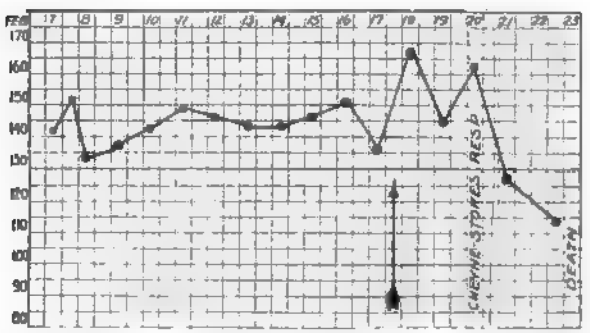


FIG. 152 — Curve of blood-pressure in a case of chronic myocarditis, showing the high blood-pressure persisting until shortly before death. (High-pressure stasis.)

This accounts for the fact that under such circumstances venesection may raise, digitalis may lower the blood-pressure, and, on the other hand, the blood-pressure may rise rather than fall as death approaches (Fig. 152).

Arrhythmia. — In many cases of chronic myocarditis the heart is irregular in both force and rhythm, especially in the later stages.

The chief types of arrhythmia observed are:

(1) Extrasystoles of auricular or more frequently of ventricular origin.

(2) Perpetual absolute arrhythmia.

The extrasystoles seem to result from the overloading of the chambers in which they arise; the perpetual arrhythmia both from the overloading of the auricle and the presence of chronic myocardial changes in the muscle strands of the intervenous area (embryonic sinus remnens). (The mechanism and significance and diagnosis of these arrhythmias have been discussed on page 75.) This irregularity in itself also exerts an unfavorable action upon the circulation. When the site at which the impulse arises is diseased, it may be impossible for this area to generate cardiac impulses in rapid succession, and hence the pulse and the circulation may remain slow in spite of the needs of the body for increased aeration.

SYMPTOMS AND SIGNS.

From the above cited cases it will be seen that the symptoms of chronic myocarditis are usually those of gradually developing cardiac weakness, progressively increasing weakness and dyspnœa, at first on exertion, later when at rest, and finally reaching the stage of orthopnœa. Palpitation is a frequent symptom, sometimes there is precordial pain, usually behind the sternum, associated with sudden dilatation of the heart. Swelling of the abdomen and often pain in the right hypochondrium are associated with the stretching of the capsule of the liver as the latter enlarges. Œdema ascending from the feet and legs, diminution in the amount of urine, at first during the day, with frequent and increased micturition at night, and later marked diminution in total urine secretion mark the later stages of broken systemic compensation

Physical signs are: cyanosis; dilatation of the venules, especially over the face; general engorgement of the larger veins, often with disappearance of the "double" venous pulse, and either total absence of the pulsation in the jugular veins or appearance of a "single" venous pulse; often irregularity of the arterial pulse, usually with presence of marked arteriosclerosis, increase in area of cardiac dulness to right or felt; occasionally a catarrhal jaundice is a marked sign of the hepatic engorgement.

The blood count often shows polycythæmia and high hæmoglobin, without change in leucocytes Blood-pressure may or may not be elevated; but in most cases it is not decreased. Pulse tracings from the radial and carotid arteries and jugular veins often show persistent absolute arrhythmia, with paralysis of the auricles, with absence of signs of organic valvular lesion. There may be a more or less transitory soft systolic murmur present at apex due to functional mitral insufficiency, but this is rarely transmitted to the axilla and often passes off during treatment. The same applies to the systolic murmur, which may be loudest over the tricuspid area. There is usually absence of diastolic murmurs except in cases in which functional pulmonary or aortic insufficiencies are suspected.

A mild bronchitis with râles and some œdema is common, especially at right base. Enlargement of the liver, with either systolic impulse

(tricuspid insufficiency) or systolic retraction (tumultuous action of the right ventricle), occurs in the later stages.

The urinary findings, cardiac symptoms, and clinical course in such cases may be very similar to those of cases which are primarily renal in origin.

Case of Chronic Myocarditis

George G., a laborer, aged 56, was admitted to Prof. J. O. Hirschfelder's wards of the City and County Hospital, San Francisco, on April 21, 1905, complaining of asthma. His father had died of dropsy. The patient had had rheumatism in 1887 and 1895, and has had to pass water during the night for some years.

Except for occasional shortness of breath he was well until two weeks before admission. He has had shortness of breath for the past two years, weakness and oedema of the feet for the past two weeks.

Physical Examination.—Patient is a fairly nourished man, face flushed and venules dilated. No marked respiratory distress. Head is of peculiar shape. Pupils equal and react to light and accommodation. No jaundice. Delicate congenital external strabismus of right eye. Eyes move well in all directions. Tongue coated. Throat clear, tonsils not enlarged, no tracheal tug. No enlargement of lymph-glands. Thorax barrel-shaped. Vocal fremitus equal except below level of tenth dorsal vertebra on right side, where it is increased. Percussion note everywhere clear except over this area, where breath sounds are distant and a few rales are heard. A few moist rales are also heard over the apices. Heart.—Diffuse but feeble impulses in sixth left interspace 15 cm. from midline, from which point cardiac dulness extends above to the upper border of the third rib and 6 cm. to the right of the midline in the fourth interspace. Heart sounds feeble and accompanied by a soft systolic murmur. Neither sound at base specially accentuated. Pulse very feeble, rapid and irregular. There is no auricular wave upon the tracing of the venous pulse and the arrhythmia is devoid of any regularity in sequence. Radial arteries are very sclerotic. No oedema of feet or legs.

Patient has some cough, raising mucopurulent sputum with large numbers of streptococci but no influenza or tubercle bacilli. Urine negative, sp. gr. 1010, no albumen, casts, or sugar.

Ordered: Soft diet. Pil. cathart. co., ii, q. n.; sol. magnesii sulphatis sat. 30 c.c. (℥j) q. 4 h. p.m.; fluidextract digitalis, 0.3 c.c. (℥v) q. 4 h., spir. glycerylis nitratis, q. ½ h., commencing with 1 gtt. and increasing 1 gtt. at each third dose until patient feels throbbing of the head or flushing of face, after which next dose is to be omitted, and subsequent doses of 1 gtt. less than the last are to be then given. Morphin. sulph., 0.008 Gm. (⅛ gr.) p. r. n. (for extreme dyspnœa).

April 25, 7.00 P.M. No change in condition. No urgent dyspnœa. Hæmoglobin 110 per cent. (Dare). Cyanosis still marked. No auricular wave in venous pulse. Heart's action still weak and irregular.

1100 c.c. of blood were then removed from right arm, after which hæmoglobin fell to 65 per cent. The right border of cardiac dulness retreated 1 cm. toward midline; upper border receded 5 cm., left border unchanged. No change in cardiac sounds nor in pulse tracing. No auricular wave in venous tracing. Blood-pressure: before venesection, 7.00 P.M., maximal 107, minimal 87, pulse-pressure 20, pulse-rate 116, pulse-pressure × pulse-rate = 2320. after venesection 8.30 P.M., maximal 112, minimal 92, pulse-pressure 20, pulse-rate 112, pulse-pressure × pulse-rate = 2240 (see chart, page 176). Cyanosis has, however, been replaced by a healthy color, and patient feels decidedly better. The improvement in this case is due entirely to relief of the over-distended right heart, partly by diminution of fluid, partly by diminution in the viscosity of the blood from the removal of so many blood-corpuscles.

The patient passed a comfortable night and for several days felt somewhat better. The course of symptoms and their relation, medication, and blood-pressure changes are shown in the chart (fig. 130,. He was bled 350 c.c.) again on May 14, with considerable benefit, and from that time his condition steadily improved.

PARALLELISM BETWEEN MANIFESTATIONS OF PRIMARY MYOCARDITIS AND PRIMARY NEPHRITIS.

The cases of chronic myocarditis with arteriosclerosis and secondary renal involvement often very closely resemble those of primary renal involvement with secondary myocarditis, since there are both cardiac and renal failure in both conditions.

The following abstracts show the close parallelism between the symptoms and signs of two such cases which in the early stages were almost exactly similar:

	Chronic myocarditis (C. B.). (Diagnosis on first admission "chronic nephritis").	Chronic nephritis (J. B.).
Illness....	Shortness of breath, palpitation, cough, swelling of abdomen and legs. Voids during night.	Shortness of breath, orthopnœa, swelling of legs.
Signs.. ...	Pale pasty color. Moist râles in chest. Heart dilated to left (15 cm.); rapid regular pulse 120; sclerotic radials. Maximal blood-pressure. 180 mm. Hg. Later, two attacks of angina pectoris, with death in the second.	Pale pasty color. Moist râles in chest. Heart dilated to left (14 cm.) and right (5 cm.). Pulse rapid and regular. Maximal blood-pressure 200 mm. Hg; later ranged from 130 to 170 mm. Hg. Fundi oculorum normal. Later, Cheyne-Stokes breathing. Delirium; headache; dulness.
Urine.....	Varying from 2000-3000 c.c. per day, with sp. gr. 1007, trace of albumen and a few hyaline casts, to less than 1700 c.c., with sp gr. 1020, large amount of albumen, and numerous hyaline casts.	Urine varied from 400 c.c., with sp. gr. 1022, 2.5 Gm. albumen per litre, and numerous hyaline and granular casts, to 2500 c.c., sp. gr. 1007, trace of albumen, and few casts.
Autopsy...	Heart hypertrophied 650 Gm., auricles dilated; intense cardiosclerosis, with some hypertrophy. Both coronary arteries diseased, left descending branch almost obliterated.	Heart dilated 350 Gm.; pale pink walls, with slight fibrosis. Coronary arteries sclerotic.
	Kidneys large, purple, with a few depressed scars and retention cysts; cortex thicker than normal; no increase in interstitial tissue; no marked nephritic changes.	Kidneys small, scarred, cortex thin; extensive epithelial degeneration with corresponding proliferation of connective tissue. Many glomeruli have undergone fibrosis.
	Adrenals—fatty degeneration of cortical cells; no hypertrophy.	

It may be almost impossible to establish differential diagnosis between two such cases early in the disease. The course of the two cases, however, showed clearly the divergence, the one toward the type of coronary sclerosis, dilated heart, precordial pain, paroxysmal dyspnœa, the other toward the uræmic, with progressive dulness, oliguria. Albuminuric retinitis did not develop in the case cited, or the diagnosis might have been simplified.

Catalase Test.—Recent studies of M. C. Winternitz indicate that in many cases at least the diagnosis may be made by a simple chemical test.

He has found that in chronic nephritis the catalase of the blood is destroyed, so that, when placed in contact with hydrogen peroxide, no oxygen is liberated; while the blood of patients with cardiac weakness splits peroxide as before. By this test he has made correct diagnosis in a number of doubtful cases. However, this difference in the catalase manifests itself only in the uræmic and preuræmic states and is of value only in distinguishing between these conditions and cases of myocardial weakness with drowsiness.

DIAGNOSIS.

In making the diagnosis it is most important to differentiate chronic myocarditis from the following conditions: (1) organic valvular heart lesions, (2) obesity, (3) primary cardiac overstrain, (4) primary chronic nephritis, (5) chronic polycythæmia (erythræmia) with enlarged spleen, (6) neurasthenia and psychasthenia, (7) chronic nephritis.

In cases of chronic myocarditis it may be extremely difficult to exclude an organic valvular disease. This is especially true of mitral insufficiency, for there is frequently a functional mitral insufficiency present with systolic murmur and horizontal dilatation of the heart to the left. While it is true that the murmur of a functional mitral insufficiency is rarely as rough as those of organic origin may become, and is as a rule not as well transmitted into the axilla, nevertheless in individual cases these differences may not be striking. Much more striking are the changes in the character of the murmur as the patient's condition improves. In organic lesions the murmur will become louder as improvement sets in, because the heart has become stronger. In functional cases, though it may become louder at first, it will vary greatly in character and in intensity, especially if the patient is made to exercise slightly. It may show a tendency to disappear altogether during recovery.

The presence of a large, slow, heaving apex beat with slow pulse and systolic murmur as well as a large slow pulse speaks in favor of organic mitral insufficiency (marked hypertrophy of the left ventricle), though a functional papillary insufficiency might persist from localized myocarditis of one of the papillary muscles in spite of the hypertrophy.

From other valvular diseases the diagnosis is comparatively easy. In occasional cases the beat of the auricle becomes audible, suggesting the presystolic rumble of mitral stenosis. Sewall[1] and occasionally blowing diastolic murmurs at the sternal margin suggest organic aortic or pulmonic insufficiency. But such dilatations of the aortic ring and conus arteriosus or cardiopulmonary murmurs are rather rare and are usually transitory.

A functional tricuspid insufficiency results so constantly from weakening of the right ventricle that it is a lesion to be included under rather than excluded from the picture of chronic myocarditis.

Primary cardiac overstrain may be excluded through the history, the trouble in the latter condition coming on suddenly in a previously healthy individual during or immediately after a severe strain, while in chronic myocarditis there is usually a more gradual onset of symptoms frequently traceable to febrile disease or intoxication.

Obesity is diagnosed from the general appearance of the patient, concomitant chronic myocarditis being excluded when the trouble seems to bear a relation to too good health rather than to disease. However, myocardial changes may be very hard to rule out.

The differentiation from chronic nephritis has been discussed above.

Chronic polycythæmia (erythræmia) with enlarged spleen may present a picture very similar to primary chronic myocarditis, and in the later stages a considerable grade of myocarditis may be present. The size and hardness of the spleen, the color, and the high blood count are the features upon which the diagnosis is made.

Neurasthenia cardiaca, neuroses, or pseudocardiac visceral disease, must be carefully excluded (see page 708). In the former the weakness when self-conscious and the strength when the mind is distracted are totally disproportionate, while the myocarditic is reminded of his weakness by the stern hunger for air.

A careful general examination should always be made to exclude cardiac weakness from enteroptosis and similar disorders that may reflexly give rise to a true cardiac weakness.

The venous pulse helps somewhat, the presence of a visible "single venous pulse" of auricular paralysis or extrasystoles suggesting myocardial change. However, these may not be conclusive. For the past year the writer has had under observation a young athlete with permanently irregular pulse and auricular paralysis and symptoms of slight cardiac weakness on exertion. There are, however, no infectious diseases nor indiscretions to account for the production of a myocarditis and, though the writer inclines toward the diagnosis of the latter condition, it seems difficult in so healthy a young person to exclude a neurotic basis.

TREATMENT.

The treatment of chronic myocarditis in the main should follow the general scheme laid down in detail in Chapters IV., V., and VI.: rest in bed during the severer stages of failure, purgation, light diet, digitalis or strophanthus in severe cases, graduated resistance exercises and Nauheim baths during convalescence, gradually increasing walks and moderate exercise before returning to every-day life. However, certain exceptions must be noted, especially in the severer forms of myocarditis. For example, digitalis only occasionally corrects an irregularity which has become relatively permanent; though it is very useful in curing the milder forms of irregularity, such as a continual bigeminal pulse or occasional ventricular extrasystoles. It is less, indeed rarely, efficient in removing the irregularities arising at the auricles. On the other hand, in dealing with the advanced grades of permanent arrhythmia with paralysis of the auricles, where there is usually advanced myofibrosis and only a few of the heart muscle-cells have survived the general atrophy, it is found that these often respond well to small doses (about half the normal), whereas a normal dose may give rise to symptoms of definite digitalis poisoning and often hasten death. This is not always to be avoided by the apparently mild routine of administering the drug in "courses," since the initial dose may be too large for the individual case. Each case must be considered for itself, with these facts constantly borne in mind. The moderate-sized initial dose or two followed by prolonged administration of very small doses, suggested by Frankel (see page 179), seems to be the safest and surest method in these cases, in order to prevent cumulative effects.

The recent introduction of single doses of strophanthin intravenously, which does not increase peripheral resistance, gives promise of great results in the future, especially in this group of cases, although its use has not yet become general enough to warrant a verdict.

As to graduated exercises, these are useful in many cases, but are distinctly contraindicated after myofibrosis has set in and dyspnœa persists while the patient is at rest. Mere arrhythmia, even with paralysis of the atria, does not contraindicate their use, but points a warning, and in many cases shows that the practitioner is treading on dangerous ground. This applies also to Nauheim and other baths. Coronary sclerosis, on the other hand, stenocardia, and severe pains down the arms furnish distinct contraindications to all exercises except such as are necessary. Even those of Schott must be carried out with the utmost precaution, and the bending exercises may well be eliminated. The walks, etc., which

terminate the treatment must be taken slowly and with the greatest precaution in avoiding fatigue.

For the stenocardiac attacks and paroxysmal dyspnœa the greatest relief is given by a pearl or two of *amyl nitrite* followed by *nitroglycerin* and *sodium nitrite*. Indeed, these drugs furnish a good deal of relief where the arteriosclerotic element is prominent.

When the blood-pressure is elevated above 140 mm. the *salt* in the food should be reduced as low as possible (see page 168).

Venesection may be of the greatest value in tiding over periods of acute dilatation, as shown in the case of G. G., even when, as in that case, it produces no change in maximal or minimal blood-pressure or pulse-rate. This case also exemplifies the fact that the venesection may often be of great benefit before acute signs of cardiac overfilling set in, and then it is to be regarded as "a stitch in time," the relief of the over-distention enabling the heart to right itself. This may be owing to the fact that the overstretched fibres are allowed to gain their optimum length, or, on the other hand, to the removal of a large number of red corpuscles from the circulatory system, thus decreasing the viscosity. It is easier to pass than to seize the moment at which a venesection would do most good.

In this every one some day or other receives his lesson. For example, the writer had a patient under his care in San Francisco who one night had a moderate degree of dyspnœa and cyanosis, though scarcely enough to cause alarm, and immediate venesection was considered. The right heart was not markedly enlarged and none of the objective signs seemed urgent. It was decided to do the venesection the next day, and the patient was given fifteen milligrams (a quarter grain) of morphine, after which he fell into a quiet sleep almost immediately. A couple of hours later he became restless and sank gradually within an hour. We had let the right moment for the venesection pass, and had masked the symptoms by the morphine.

Dangers from Morphine.—Another danger due to *morphine* lies in the danger of habituation (see page 149), and the further danger that in order to get it the patient will *simulate* a paroxysm of dyspnœa and actually make himself sick or even endanger his life by the effort entailed in doing so. Several patients whom the writer has gradually broken of their morphine habit confessed to having done so, even though they knew at the time that the simulation of dyspnœa made them feel worse.

THROMBI IN THE CARDIAC CHAMBERS

When the circulation is slowed, and especially when one of the cardiac chambers empties itself insufficiently, large clots are liable to form along its wall (mural thrombi). This occurs especially in those portions which are away from the axial stream, such as the recesses between the trabeculæ carneæ and behind the papillary muscles, and also out in the tip of the auricular appendages.

Thrombosis within the left auricle occurs quite frequently in *mitral stenosis*, especially when the blood stagnates there during periods of overstrain. These thrombi if fresh sometimes break loose to form emboli (page 151). Sometimes the clot loosened from the auricular appendix is so large that it cannot pass through the auriculoventricular orifice, but plugs the latter entirely, producing sudden death. When the clot remains adherent to the wall for some time, more or less organization goes on. Thrombi which

adhere to the wall by a few strands of newly formed connective tissue are of every-day occurrence, and constitute the classical sign for differentiation between intra-vitam and post-mortem thrombi. In older thrombi the organization is more complete, so that a thrombus mass may adhere to the cardiac wall by a pedicle of fibrous tissue. It is quite possible that in some cases these thrombi vibrate to and fro and cause extrasystoles by striking against the walls of the heart, just as occurred in Cameron's air-bubble experiment (quoted on page 71). In several cases such masses have been known to act as a ball-valve at the mitral orifice, giving rise to signs of mitral stenosis.

The symptoms and signs given by such thrombi are, however, very obscure. The fact that they usually arise during the course of a cardiac failure adds to the complexity of the clinical picture, and the diagnosis can rarely be made until embolism sets in. In one case of mitral stenosis recently seen by the writer, in which the whole descending abdominal aorta was suddenly plugged by an embolus and gangrene of both lower extremities set in, the diagnosis of a clot within the heart was warrantable. Such cases are, however, rare, and the diagnosis is then made after the harm has been done.

TUBERCULOSIS OF THE HEART.

In spite of the great frequency with which tuberculosis affects the lungs, pleura, and pericardium, independent affection of the myocardium, endocardium, and valves is quite infrequent.[1] Thus Willigk found only 2 cases of tubercle of the myocardium in 1845 autopsies on persons with tuberculosis. Other observers confirm this view of its rarity.

Pathologically the lesions in tuberculosis of the myocardium resemble those of tubercles elsewhere; they are somewhat more common in acute miliary tuberculosis than in the chronic form, but in the latter are larger in size. The most common cardiac lesion of tuberculosis is, however, neither miliary nor large solitary tubercles, but a fatty degeneration of the myocardium, due in part to the anæmia and in part to the toxins secreted by the bacilli.

The effect of the tuberculous lesions upon the circulation is usually masked by the general cardiac weakness due to the intoxication and anæmia, and, as v. Leyden states, does not present any characteristic features. It is almost impossible to diagnose clinically, for the symptoms and signs are quite independent of the tubercle. Often, as in Pollak's case, a man of 65 who had a large tubercle in the wall of the auricle, there are no signs whatever, even of cardiac weakness. V. Tabora and Tilp report a case in which a systolic murmur was heard over the apex, but this, of course, presents nothing characteristic and might well have been due to the accompanying weakness of the myocardium or papillary muscles. Indeed, as Romberg states, tuberculosis of the myocardium interests the pathologist rather than the clinician.

SYPHILIS OF THE MYOCARDIUM.

Syphilitic affection of the heart is more frequent and presents a somewhat more definite picture than tuberculosis. The most common form in which syphilis affects the heart is the sclerotic lesion of the aortic valves (see page 361), though in this case the pathological process originates in the aorta rather than in the myocardium.

Grassmann has called attention to the frequency with which signs of severe cardiac weakness occur during the secondary stage of syphilis, accidental or functional systolic murmurs being present in 10 per cent. of his cases. Dilatation especially of the right heart, was common, as well as alterations of rhythm—sometimes arrhythmia, sometimes bradycardia, sometimes tachycardia. Precordial pain and anginal attacks were frequent. The blood pressure was usually low, as was also the hæmoglobin. It is not impossible that the major rôle in many of these cardiac manifestations is played by the anæmia and the fever rather than by spirochætæ pallidæ within the heart muscle; but the presence of tertiary myocardial lesions demonstrates that the latter play an important part. The diagnosis is based upon the above-enumerated symptoms arising during the secondary stage. Treatment should, of course, be vigorous, and as a preliminary measure the patient should be kept in bed until all cardiac weakness has passed. If the symptoms do not rapidly subside, a few doses of digitalis or strophanthus may be

[1] Tuberculous endocarditis is discussed on page 303.

given. Indeed, a few small doses of one of these drugs may well be given to relieve promptly the dilatation and thus to forestall the danger that may lurk in an œdematous heart muscle (see page 235).

Cardiac lesions are rather common in congenital syphilis, though this is not true of typical gummata. Thus Mraček found myocardial changes (acute myocarditis with patches of perivascular infiltration of mononuclear cells) present in 24 out of 150 autopsies upon syphilitic foundlings, but gummata in only 4. The non-gummatous changes are well described by I. Adler as infiltrations of mononuclear cells about the blood-vessels and in the connective-tissue septa between the muscle-fibres. The striking feature is early typical chronic endarteritis with thickening of the intima, destruction of the elastica interna. This is often accompanied by hemorrhages into and about the vessel wall. Clinically hereditary lues of the myocardium probably coöperates with the other syphilitic lesions in bringing about the death of the child, but the importance of its rôle cannot be judged, since it is rarely if ever the only luetic lesion present.

The tertiary myocardial lesions of adults are fairly common. The lesions in 60 cases collected by Mraček showed the following distribution: gummatous myocarditis, 10; fibrous myocarditis, 9; gummatous and fibrous, 8; endocarditis, 2; coronary arteries alone, 3; pericardium alone, 1; myocardium and pericardium, 15; pericardium, myocardium, and endocardium, 1; myocardium and coronary arteries, 1; all parts of the heart, 6; cardiac ganglia, 4.

Judging by the number of cases of Adams-Stokes syndrome due to lues (see page 471), the intraventricular septum seems to be a rather frequent site for the lesions. Excepting such lesions as are so situated that they give rise to heart-block or to the Adams-Stokes syndrome, the syphilitic lesions of the myocardium rarely give distinct manifestations. A general myocardial weakness, shortness of breath, dilatation with or without exertion in persons who have had lues (especially with other visceral involvement) is suggestive evidence of fibrous luetic myocarditis with or without gumma. The latter can rarely if ever be diagnosed. Huchard and Fiessinger report a case in which dyspnœa set in suddenly 15 days before death, due to the growth of a gumma involving the tricuspid valve, but even in such a case the data are too uncertain to permit a definite clinical diagnosis. A positive Wassermann reaction, which Collins and Sachs and W Longcope have found so useful in the diagnosis of luetic aortic insufficiency, is of less value in the diagnosis of luetic myocarditis, since the evidences of myocarditis are in themselves less definite. However, in cases of chronic myocardial weakness in which lues is suspected, the presence of a positive Wassermann reaction renders a vigorous administration of mercurial inunctions or hypodermic injection of mercurial salts, as well as vigorous doses of potassium iodide, highly advisable In occasional cases it may be possible to secure a considerable and permanent improvement by vigorous antiluetic treatment, even when the Adams-Stokes syndrome is present; but it must not be forgotten that the cardiac infiltrations are among the most stubborn of all luetic lesions.

TUMORS OF THE HEART.

Primary tumors of the heart are so rare that in 3000 consecutive autopsies at Nürnburg Thorel did not encounter a single one, and Hektoen, who reported three cases in 1893, states that reports of only 110 cases of cardiac tumors were to be found in the Index Catalogue of the Surgeon-General's Library, and most of these were secondary.

Primary Tumors.—Bertheson was able to collect 28 primary tumors of the following types: sarcoma 9; myxoma 7; fibroma 6; carcinoma 3; lipoma 2; cystoma 1. Link (1909) has recently collected the data of 91 cases; 61 of these were as follows: carcinoma 7; fibroma 7; myoma 5; lipoma 8; sarcoma 13; myxoma 18; rhabdomyoma 1; teratoma 1; papilloma 1. In addition to these Knox and Schorer and Wolbach have collected 12 cases of rhabdomyoma; 6 of which were associated with other malformations, especially cerebral sclerosis and hydrocephalus.

Hektoen calls attention to the fact that the heart, and hence also its primary tumors, are of mesoblastic origin; which accounts for the relative rarity of primary carcinomata and the preponderance of sarcomata. Thorel believes that many of the fibromata found represent merely old organized thrombi clinging to the heart wall, and believes that many of the (relatively frequent) myxomata represent merely degenerating forms of

such thrombi. The lipomata he regards merely as abnormally large pockets of epicardial or intramural fat rather than as true tumors.

As regards site, Lauk found in right auricle 10, left auricle 24, right ventricle 14; left ventricle 8, valves 16, interauricular septum 2.

Metastatic Involvement of the Heart. Secondary neoplasms affecting the heart are somewhat more common, and scarcely any pathologist of experience has failed to meet with them, especially in cases with multiple metastasis. Of the metastatic neoplasms carcinomata are the most frequent. Thorel encountered 6 instances in his 3000 autopsies, the primary sites being uterus 2, rectum 1, gall-bladder 1, kidney 1, lung 1.

Geipel stated that in a series of 16 cases of carcinoma of the œsophagus 6 gave metastases to the heart, but this is an unusually high percentage and Thorel from his experience does not regard such œsophageal tumors as especially liable to cardiac metastases.

Clinically the presence of a tumor in the heart in itself exerts little influence, unless, as in Luce's case of sarcoma, it presses upon the auriculoventricular bundle and produces heart-block, or it is so situated as to produce either stenosis or regurgitation at a valvular orifice. The benign tumors exert little or no effect upon the force or rhythm of the heart, the malignant tumors give rise merely to signs of cachexia in which the cardiac weakness seems incidental rather than primary. In cases of generalized carcinosis and sarcomatosis, those in which the metastatic nodules are most common, the cachectic myocardial weakness is still more intense whether the tumors affect the heart or not. The accidental finding of a loud harsh murmur suddenly developing and progressing with the metastasis elsewhere in the body is very suggestive, but this is rarely encountered.

In some cases in which the tumors are superficial, pericarditis may set in. *Effusion, especially blood-stained,* is rather common under these conditions and the signs of the latter may be the first and only sign of the condition.

In 1905 the writer aspirated a pericardial exudate which contained 10 per cent. of hæmoglobin and some methæmoglobin. The patient died the next day and autopsy revealed carcinomatous masses in the myocardium wall and pericardium which were metastases from a very small primary carcinoma of the bronchus quite unsuspected during life. The finding of tumor cells in such an exudate would, of course, give the diagnosis.

Tumors of the heart, even if diagnosed, would, of course, be inoperable.

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X.

ARTERIOSCLEROSIS.

NORMAL CHANGES IN THE ARTERIES DURING LIFE.

A certain degree of progressive change in the walls of the arteries occurs normally throughout life, and is therefore not to be considered pathological. The condition of the arteries normal to a man of thirty would be thoroughly abnormal in a child, and those normal for a man of seventy would in turn be regarded as abnormal in a man of forty.

Thus, Thayer and Fabyan state that "at birth the artery (radial) is delicate, translucent, extremely thin, and collapsing. The surface on opening is perfectly smooth. The intima consists of a single endothelial layer lying directly on the surface of a deeply undulating elastica interna The media, which consists of transversely arranged smooth muscle-fibres with rather large vesicular nuclei, has a depth of seven to eight layers of cells. Connective tissue, if present in the intima and media, is extremely scanty, none being revealed by the Mallory or Van Gieson stains. There is, however, a relatively large amount of elastic tissue which appears on cross section as very thin, parallel, slightly wavy lines. The elastica externa is neither as coarse nor as deeply undulating as the interna.

"The adventitia, considerably thicker than the media, consists of compact connective-tissue fibres with relatively large nuclei. The elastic fibres are fairly numerous.

"By the middle of the first decade, the intima has become thicker owing to the appearance of a fresh layer of elastica interna, while more muscle-fibres appear in the media.

"10-20 years. Walls of the vessel become thicker but still collapsed. Intima and media thicker, the elastic tissue being relatively less marked

"21-40 years. Slight further general thickening of intima and media. A second elastic layer appears in the intima. In the media the connective tissue begins to be demonstrable by Van Gieson's stain

"41-50 years. Decided change. Lumen of the vessel remains open Areas of calcification in the deep layers of the intima are frequent. The media reaches its maximum thickness. There is a good deal of connective tissue.

"After the fifth decade there is a progressive increase in the thickness of the intima . . . . and a diffuse connective-tissue thickening becomes the common type. . . . . The media after the fifth decade becomes on the whole rather thinner; there is a marked increase in the connective tissue.

"Calcification in the deep layers of the intima becomes more common with age, four out of five cases in the eighth and ninth decade showing this change."

PATHOLOGICAL ANATOMY.

Theoretical Considerations. — Pathologically, arteriosclerosis is characterized by the occurrence of changes in and thickening of the intima, which was supposed by Rokitansky to be due to the depositing of cells directly from the blood stream; by Virchow to be a true inflammatory hyperplasia as the result of some "formative stimulus"; and by Thoma to be a compensatory thickening of the wall in order to diminish the lumen of the vessel after the stretching which occurred under the increased blood-pressure with which it was usually associated. Jores, on the other hand, regards this as a true hyperplasia resulting from

the high blood-pressure but independent of the lumen of the vessel, returning to a certain degree to the view of Virchow. These observers considered the changes in the intima as primary, and tended rather to neglect the second important change which characterizes arteriosclerosis, namely inflammatory changes within the media.

On the other hand, Koster and his pupils called attention to the importance of degenerative and calcareous changes in the media and adventitia as well as in the intima. Koster studied the inflammatory process very carefully by means of serial sections and injected specimens, and claimed that the arteriosclerotic lesion always took its origin in the adventitia as an infiltration surrounding the vasa vasorum like a sleeve. This infiltration followed the vasa vasorum into the media. Koster found that in the normal artery the vasa vasorum do not pass deeper than the outer third of the media, though in certain arteries (notably those of the brain and the lungs) there was a fine capillary network penetrating the deeper layers of the media as well and spreading along the medial surface of the elastica interna.

Changes in Vasa Vasorum. — This view is confirmed by v. Ebner (in Kolliker's Handbuch der Gewebelehre), who states that "the media of the larger arteries and veins, according to the consensus of opinion of many authors, contains blood-vessels, though in small numbers and only in the external layers; whereas the inner layers of the media and the intima seem to be always free from vessels (in the ox the wall of the vena cava is richly supplied with vessels even down to the intima).

The infiltration about the vasa vasorum follows these paths, setting up areas of infiltration, necrosis, and calcification in the smooth muscle and elastic fibres of the media. When it penetrates to the elastica interna a small area of this is first injured, the inflammation acts as a stimulus, and hyperplasia of the intima sets in. The intima becomes thickened until its cells undergo spontaneous fatty degeneration, after which they either calcify or the capillary network penetrates through the elastica interna and a true process of organization and proliferation of connective tissue goes on.

Koster admits that it is possible that the degenerative and hyperplastic changes in the intima may go on without the entrance of blood-vessels, as do those seen in inflammations of the cornea, but he states that if the lesions are followed in serial sections there is almost always a demonstrable continuity between the patches of endarteritis, mesarteritis, and periarteritis.

The number and size of the vasa vasorum and the richness of the capillary network are always increased in arteriosclerosis and in phlebosclerosis. He states that endarteritis occurs only in arteries that have vasa vasorum, that is, in the larger arteries and in the smaller arteries of the brain and the lungs.

Koster's version is extremely fascinating, especially since it presents a simple explanation of a complex picture, and, on the other hand, presents a clear analogy with the processes involved in myocarditis, endocarditis, and other lesions. As far as the media and adventitia are concerned his findings have been confirmed by Ophuls, whose careful study constitutes one of the most important and clearest of the recent contributions to the subject. Ophuls, however, was unable to demonstrate any constant relation between lesions in the media and those in the intima and believes that they are produced independently though from the same general cause.

He states that "anatomically arteriosclerosis of the aorta is a unit. It is a chronic inflammatory process of the vessel wall which attacks all the coats simultaneously, which as a rule first produces changes in the intima and adventitia." He believes therefore that, as Koster suggested, the changes in the intima begin as parenchymatous changes without the presence of blood-vessels, like the inflammations within the cornea.

CLASSIFICATION OF ARTERIOSCLEROTIC LESIONS.

Following this view in adopting a pathological classification, one might distinguish the following groups of arteriosclerotic lesions, dependent upon the arterial coat most affected and the distribution of the lesions within that coat.

FIG. 153.—Various types of arteriosclerotic lesions. (Schematic.)

I. Adventitia chiefly affected (no weakening of arterial wall).
   1. Localized or nodular infiltrations (periarteritis nodosa).
   2. Diffuse infiltrations about the vasa vasorum (causing uniformly thickened arteries which give the sensation of thick rubber tubing).

II. Lesions in the media predominate (with weakening of the arterial wall), especially common in syphilis.
   1. Localized necrosis of elastic tissue with calcification (atheroma) (papestem or "goose-neck" arteries, Monckeberg's arteriosclerosis, experimental toxic arteriosclerosis).
   2. Diffuse or patchy medial fibrosis with more or less calcification, often leading to aneurism.

III. Changes in the intima predominate (with no weakening of arterial wall).
   1. Hyperplasia of intima with fatty degeneration at its centre (acute aortitis): (a) without ulceration, with calcification of the areas of fatty degeneration (atheromatous plaque or "ulcer"); "endarteritis deformans" (Lotzbeck).
   2. Simple hyperplasia of intima (diffuse endarteritis) with increase of elastic fibres, finally leading to
   3. Obliterative endarteritis, in which the process is still more chronic and intense and capillaries enter from the vasa vasorum.

According to Weiss and v. Winiwarter, and later Boerger, this last is to be sharply differentiated from thrombangiitis obliterans, in which intravascular coagulation precedes or is independent of the change in the intima, at which the lumen of the vessel finally becomes obliterated by secondarily forming granulation tissue devoid of elastic fibres and arising from about the newly formed ends of the vasa vasorum.

Periarteritis nodosa.—Kussmaul and Maier (supra-arterial fibroid nodules), one of the rarer forms of arteriosclerosis, is produced by the formation of small areas of nodular infiltrations in the adventitia and gives the vessel a nodular appearance and consistency. It is almost always closely associated with inflammatory changes in the media and a local proliferative endarteritis (Ziegler).

Diffuse Periarteritis.—The diffuse thickening of the adventitia (periarteritis) is more common, occurring about the arteries of the brain, about the coronary arteries in myocarditis, and in many other organs in subacute inflammatory processes. In the radial and other large arteries it seems to be quite common. The uniformly thickened arteries of leathery consistency which are so commonly met with in young or middle-aged persons who do hard work seem to be of this type, though the fact has not yet been settled with definiteness. Whether such changes may be transitory or are always permanent has not been definitely settled. The boy of six cited below (page 259) may perhaps represent such a case.

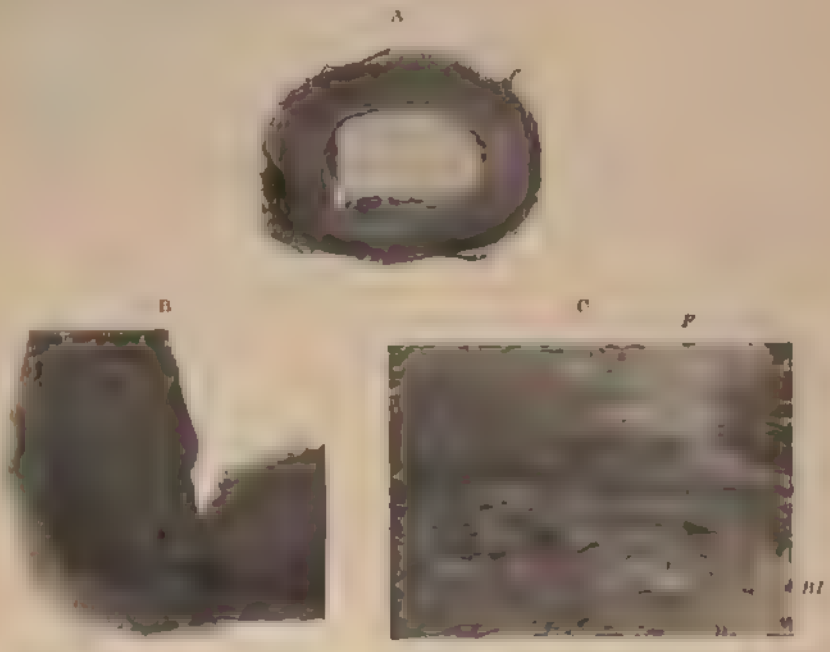

FIG. 154. Cross section of a radial artery showing arteriosclerotic changes in the media. Photomicrographs by Dr. C. S. Bond. A. Cross section of the entire artery, low power. B. Lower, oft-hand corner of the same specimen, higher magnification showing destruction of the elastica interna and thickening of the intima. C. Other specimen showing destruction of the elastic fibres, F, of the media with proliferation of white fibrous tissue, F, and blood vessels, BI. Kindness of Prof. W. Opitz.

Medial Changes (Mesarteritis). The rigid "pipe-stem" or "goose-neck" radial arteries often met with in very old persons are formed by the presence of areas of degeneration and calcification within the tunica media. This condition occurring without any changes in the intima has been described in man by Monckeberg, and represents the type of arteriosclerosis or arterionecrosis produced experimentally in animals with bacterial toxins, acids, adrenalin, and alkaloids.

Calcification. According to Klotz, Wells, and Baldauf, the process of calcification seems to go on in the following way. As a result of the inflammatory changes, the muscle cells degenerate and the lecithins become split up into fatty acids, glycerophosphic acid, and cholin, causing the appearance of a fatty degeneration. The calcium and magnesium in the serum then enter into combination with the phosphoric acid and are precipitated to

form calcareous plaques and granules in the media. Klotz believes from histo-chemical evidence that there is intermediate or concomitant formation of calcium soaps, but Wells and Baldauf have not found soaps on chemical analyses. Baldauf and also Selig find that most of the calcium is in the form of phosphate and sulphate, little as carbonate. The ash from arteriosclerotic plaques contained CaO 53·84 per cent., Fe_2O_3 0·25 per cent., Na_2O 0·72 per cent., K only traces, P_2O_5 40·19 per cent., SO_3 0·43 per cent., Cl trace, F negative, CO_2 traces (Selig).

Klotz finds that the calcium is by no means always deposited in the patches of atheroma, but exists also as rows of fine granules between the muscle-fibres. In this condition it gives no macroscopic evidence of its presence, and merely causes a slight increase in the rigidity of the artery. When the necrosis of the arterial wall proceeds more slowly, the phosphoric acid or glycerophosphoric acid derived from the lecithin is removed by the blood-vessels (capillaries of the vasa vasorum), which enter the diseased area of the media, and the injured elastic tissue is replaced by fibrous tissue without the deposition of calcium.

Fig. 156.—Arteriosclerosis of [illegible] showing atheromatous plaques.

Whether the calcium is deposited or not, the area of diseased media constitutes a weakened portion of the wall and is the lesion which in the large arteries is particularly responsible for aneurism formation (see also page 521). It has been claimed by some writers (Heiberg, Heller, and others) that this lesion was confined to luetic cases, but both clinical and experimental data show that it is due to non-luetic lesions about as frequently as are any of the other lesions of arteriosclerosis (Ophüls).

Intimal Thickening.—The lesion which Virchow, Thoma, and many writers have regarded as the fundamental one in arteriosclerosis is thickening of the intima. Virchow believed that it arose as an inflammatory hyperplasia resulting from some "formative stimulus" within the blood stream. Thoma believed that this stimulus was the mechanical factor of high blood-pressure, and that the thickening of the intima represented a compensatory hypertrophy to prevent aneurismal dilatation, but Ophüls has shown that, in marked contrast to the area of medial disease, there are no bulgings of aortic wall at the areas of intimal atheroma, even when the artery is distended under a pressure of 160 mm. Hg.

The "formative stimulus" is probably not mechanical but chemical, perhaps the same as those which have been shown experimentally to give rise to arteriosclerosis in small animals. Under the influence of these stimuli the intimal layers undergo hyperplasia, with increase of both fibrous tissue and elastic fibres (Fig. 156). Since, as Koster has shown, there are no blood-vessels, but only lymph spaces or lacunæ, the hyperplasia soon reaches its limit, and under the further influence of the toxic agent the cells at the centre undergo "fatty degeneration" from ischæmia. Such areas present at first a translucent appearance and are known as areas of

"acute aortitis." Later calcium salts are usually deposited (as described above), and they become converted into calcified plaques of atheroma or atheromatous ulcers. When the process is more chronic there is usually a wandering in of capillaries from the media after the manner described by Koster, and under the influence of the improved nutrition the intimal hyperplasia may go on at an increased rate even to the obliteration of the lumen (endarteritis obliterans). Thromboangitis obliterans will be considered in Chapter IX.

Unity of Arteriosclerotic Processes. — Although a large number of writers attempt to put each case into one or the other of these groups, Ophuls has shown, by a careful complete study of seventy consecutive unselected cases, that such divisions are based upon unessential differences

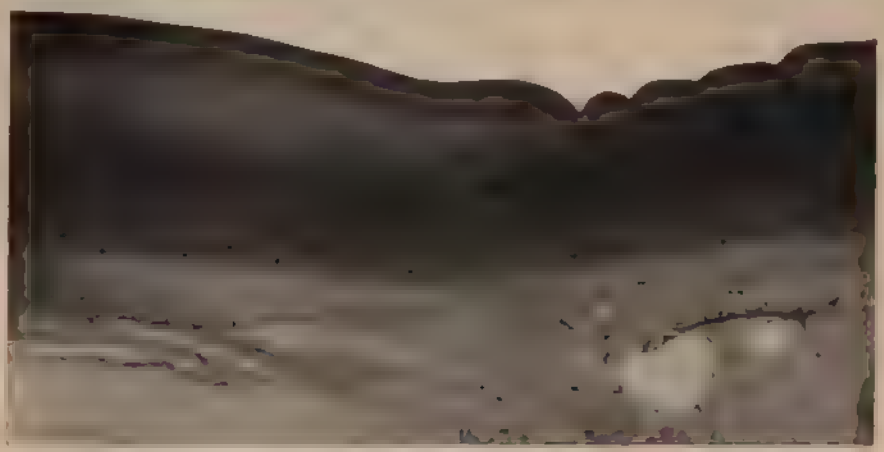

FIG. 156. — Atheromatous plaque, showing the changes in the intima. (Photomicrograph by Dr. Charles S. Bond.)

and that, as a matter of fact, any or most of the forms may arise in the same case. This division was attempted especially by Heiberg, Heller, and his pupils, who believed that mesarteritis, particularly when it attacked the first part of the aorta and the ascending arch, was characteristic of syphilitic disease. While it is quite true that syphilis may give rise to a mesarteritis, and occasionally even to the formation of miliary gummata in the adventitia,[1] nevertheless these lesions are far from characteristic, and very similar non-luetic structures occur about thrombosed vasa vasorum. Moreover, Ophuls was unable to find any difference in the distribution of luetic and non-luetic arteriosclerotic lesions in the seventy cases of his series.

ETIOLOGY.

The most important etiological factors in the production of arteriosclerosis in man are age, hard work, alcohol, syphilis, and the more acute

[1] Some writers claim to have found the spirochæte pallida in these lesions by means of the rather questionable Levaditi's silver nitrate method, though Ritter, Buerger, and many others have failed in spite of painstaking search in many cases. However, Collins and Sachs Longcope and Clough and Guthrie at the Johns Hopkins Hospital have been able to diagnose luetic arteriosclerosis during life by the Wassermann reaction.

infectious diseases, especially typhoid fever. The relative frequency of these causal factors, as indicated by the palpability of the radial artery in 4000 consecutive cases admitted to the Johns Hopkins Hospital, has been made the subject of a careful study by Thayer and Brush.

These observers found palpable arteries in the following percentage of the patients under fifty years who had been subject to various etiological factors:

After scarlatina, radials palpable in	16 4 per cent.
No causal factor, radials palpable in	16 5 per cent.
Pneumonia, radials palpable in	17 per cent.
Diphtheria, radials palpable in	17 per cent.
Malaria, radials palpable in	20 per cent.
Typhoid fever, radials palpable in	26 per cent.
Rheumatism, radials palpable in	31 per cent.
Alcohol, radials palpable in	46 8 per cent.
Hard work, radials palpable in	57 5 per cent.

Richard Cabot takes exception to these findings of the high frequency of arteriosclerosis after alcohol basing his conclusions upon autopsies of dipsomaniacs under fifty in whom he says arteriosclerosis was not present in more than twenty per cent. His exceptions to Thayer's findings are however somewhat against the general consensus of opinion, as well as against the experimental evidence of Aubertin, who produced arteriosclerosis and cardiac hypertrophy in rabbits by the injection of alcohol. On the other hand, Cabot is supported by Fahr, who performed 400 autopsies on habitual drunkards dying at the Harbor Hospital of Hamburg and found arteriosclerotic changes no more common than in abstemious individuals, occurring in 95 cases, 82 of whom were over 40 years of age. Only 7 drunkards in his series died before 40 from causes referable to arteriosclerosis. Similar changes existed in only six other patients under 40. Unlike Aubertin, Fahr was unable to produce arteriosclerosis in rabbits by administration of alcohol for over two years. From this it would appear that the evil effects of alcohol have been considerably exaggerated, at least as far as the arteries are concerned. It must be borne in mind that indulgence in a certain amount of alcohol is almost universal especially in those persons who do hard work, hence it is extremely difficult to segregate these factors in any large number of cases. If, for example a patient has had typhoid fever, has used alcohol and has done hard work, it is not logical to enter his name into each of the three columns for it is not possible to determine which of the factors is the most important.

Fortunately however, for the decision of these doubtful points the experiments of Pic and Bonnamour (l. c.) upon experimental adrenalin arteriosclerosis have shown that where *two factors are acting together*, arteriosclerosis may be produced in conditions in which it could not be brought about by one of them alone. Thus tuberculosis + adrenalin yielded arteriosclerosis in young rabbits which would not have shown arteriosclerosis after adrenalin alone, and there is no doubt that the same is true in man.

Syphilis is a most important factor, especially in the arteriosclerosis which occurs below the age of thirty-five. As stated above, it was supposed by Heiberg, Heller, and their pupils that luetic arteritis assumed a definite type, the media, the adventitia, and especially the vasa vasorum showing considerable small round-celled infiltration, but, although it is possible that the media and adventitia are attacked more constantly than in other forms, this form is not to be regarded as specific. The tendency to form lesions above the semilunar valves and along the ascending aorta is by no means confined to arteritis of luetic origin (Ophuls), though extremely frequent in the latter (Osler, Collins and Sachs, Longcope). *A positive Wassermann reaction is often obtained in cases of luetic aortitis in which there are no other active luetic processes.*

Lead poisoning (especially chronic plumbism) and gout are important etiological factors, as is also chronic nephritis. Overeating is thought to play an important rôle, especially when the diet is rich in meats, sweetbreads, livers, kidneys, etc., in other words, in purin bodies and in kreatin. The exact rôle of these substances has not been carefully studied, although Croftan found that long-continued injection of 0.5 to 5.0 mg. xanthin into rabbits caused a rise of forty millimetres in blood-pressure, as well as sclerotic changes at least in the renal arteries. (He does not describe the condition of the other arteries.) From the stand-point of both blood-pressure and gaseous metabolism it has been shown that the digestion of large meals materially increased the work of the body, producing thereby an effect not dissimilar to that of hard physical exercise (increase in pulse-pressure, increase in pulse-rate, increase in CO_2 output) (effect of large meal, after Erlanger and Hooker). It is therefore quite natural that overeating should rank with hard work as a main cause of arteriosclerosis, but the exact extent of its occurrence is more difficult to determine in a large series of cases than in an individual case in private practice.

Lastly, and still more important in the etiology of arteriosclerosis, are age and heredity (Israel).

Thus, Osler states that "entire families sometimes show this tendency to early arteriosclerosis, a tendency which cannot be explained in any other way than that in the make-up of the machine bad material was used for the tubing." This is especially true as regards alcoholism, as has been shown in a recent statistical study by Emerson, who found that this factor was of more importance than the drinking of alcohol by the individual himself in determining arteriosclerosis and longevity, and that an alcoholic ancestry was very frequently followed by a generation with a tendency to early arteriosclerosis.

Experimental Arterionecrosis in Animals. A most interesting side light upon the genesis of arteriosclerosis has been thrown by attempts to produce it experimentally in animals, especially in rabbits and guinea-pigs. The lesions which have been produced cannot be termed true arteriosclerosis like that seen in man, but are confined to the media and adventitia, the intima always remaining clear. The reason for this is not evident. Even the possibility that in these small animals the blood supply of the arterial wall is different from that in man, and that owing to this difference lesions occur most readily in the media, does not hold, since Ophuls has demonstrated the occurrence of spontaneous endarteritis in rabbits. The experimental and clinical conditions seem to be closely analogous, but it is not possible to draw an absolute parallelism between them.

Gilbert and Lion have been able to produce arteriosclerosis experimentally in animals by the injection of bacterial toxins, and this has been continued by Klotz. This fact is of great importance not only from the stand-point of experimental arteriosclerosis, but also because it establishes the importance of bacterial disease in the etiology of arteriosclerosis met with clinically.

The earliest observation of arteriosclerosis brought about by toxic action of organic compounds, and one which establishes beyond doubt the deleterious action of tobacco upon the arteries, is that of Isaac Adler, demonstrating sclerosis in the smaller peripheral arteries of rabbits as a result of feeding them with infusions of tobacco. Bovet confirmed these results by giving infusions of tobacco by stomach tube, and obtained atheromatous plaques or thickening at the base of the aorta in ten out of sixteen rabbits, while Baylac obtained sclerosis in each of eight rabbits to which tobacco infusion was injected either intravenously or subcutaneously. Jaworsky and later W. F. Lee have confirmed it in rabbits made to inhale tobacco smoke. From Baylac's experiments it would appear that

in general the liability to occurrence bears some relation to the channel by which it enters the body. This may explain the very marked action of tobacco inhaled and entering the heart directly from the pulmonary circulation in smokers, as compared with the somewhat milder effects of chewing tobacco, under which condition the nicotine passes through and is perhaps somewhat attenuated in the liver before entering the systemic circulation, and has still to pass through the vena cavæ, right heart, and pulmonary circulation before reaching the coronary circulation. In smoking, however, the nicotine enters through the lungs and strikes its first blow at the coronary arteries and base of the aorta, where the elastic fibres are under the greatest tension and hence most liable to degeneration. It is, therefore, easy to understand why smoking of heavy cigars should be one of the most potent factors in the etiology of arteriosclerosis and coronary sclerosis.

An almost new era in the study of arteriosclerosis was, however, introduced by the discovery of Josué that the repeated intravenous injection of adrenalin into rabbits brought about sclerosis and calcification in the aorta within a few weeks. This was very soon confirmed by W. Erb, Jr., who produced the lesions in a large number of animals and demonstrated the considerable uniformity with which such lesions followed the injections. Similar results have been obtained in rabbits by Fischer by the intravenous injection of a very large number of substances, hydrochloric acid, phosphoric acid, lactic acid, calcium phosphate, chloralamide, mercuric chloride, trypsin, diuretin, and physiological salt solution, so that the effect can scarcely be considered as specific for adrenalin.[1]

On the other hand, Pic and Bonnamour, as well as Adler and Hensel, have called attention to the fact that in none of the series of experiments published did more than a certain number of the animals injected show lesions, and in a very large series the latter showed that it was practically impossible to produce arteriosclerosis in rabbits by these poisons until they had attained a certain age. After that age arteriosclerosis occasionally occurred spontaneously, but could be brought on with considerable frequency by the injection of toxic substances. As stated above, Pic and Bonnamour have, however, been able to produce it in young animals whose vitality was diminished by tuberculosis, etc., indicating that disease may be an accessory factor in diminishing the resistance of the arteries to toxic influences which ordinarily leave no traces. This carries the clinical corollary that persons liable to arteriosclerotic changes should particularly avoid all contributing factors (alcohol, tobacco, hard work, etc.) for some time after infectious diseases.

It is quite remarkable that Pearce and Baldauf, as well as other investigators, report that they have been able to produce arteriosclerosis, and that Josué claims to have produced permanent elevation of blood-pressure in rabbits by a single injection of adrenalin, since Fleisher and Loeb failed to do so in a large series of experiments in which such injections did produce severe myocarditis.

Mechanism Producing Experimental Arteriosclerosis. The mechanism by which arteriosclerosis is produced has been the object of considerable study. In the case of adrenalin at least, Erb believes that a spasm of the vasa vasorum takes place, bringing about an insufficient blood-supply to the coats of the vessels and thereby ischæmic degeneration of the latter, especially of the tunica media. This view was also shared by Pearce and Stanton and other observers, but Fleisher and Loeb have shown that considerable areas of aorta may be kept ischæmic by compression without producing arteriosclerosis. The factor must, therefore, be toxic. It is possible that in some cases with high blood-pressure actual rupture of the weakened elastic fibres takes place, which serves as a centre for areas of necrosis. W. H. Harvey has shown that if bits of excised aorta are filled with agar under various pressures and then transplanted into subcutaneous tissue, those under tension degenerate more rapidly. The same is probably true of the fibres within the artery. Moreover, Josué has shown that repeated injections of adrenalin in the rabbit are followed by permanent rise in blood-pressure. An increase in blood-pressure is indeed the rule in arteriosclerosis, although as Hasenfeld has pointed out, it occurs only in persons whose sclerosis involves the splanchnic arteries. Neither increase in blood-pressure nor hypertrophy of the heart necessarily occurs in patients where these vessels are not involved. The

[1] A summary of the recent literature upon this point will be found in the papers of Saltykow, Adler, and Benda.

reason for this may be that the cutting down of the circulation of so large an area as the splanchnic region in itself increases the resistance to blood flow and thereby raises pressure. There is also no doubt that, besides the single artery involved in the sclerosis, the latter is often the result of prolonged vasomotor spasm in the femoral artery etc. On the other hand, such spasm may be transitory and be accompanied by temporary rise of blood-pressure and sensory phenomena which cause the syndromes described by Pal as vasomotor crises (see page 270). Aubertin, Vaques, Wiesel, and others have found hyperplasia of the adrenals present in many experimental and clinical conditions in which hypertrophy of the heart and high blood-pressure are present. It therefore seems quite possible, in the light of these findings, that hypertrophy of the heart and arteriosclerosis may often be the result of a *hypersecretion of adrenalin*, perhaps also of some other internal secretions. Why this should be associated with splanchnic arteriosclerosis is easy to see. The latter condition tends to diminish the circulation through the abdominal viscera, and more blood is thus shunted through the adrenal arteries which lie just above the mesenteries, thus bringing about an increase in adrenal secretion.

It may be added that Bayer, in Krehl's clinic, has shown that sometimes the high blood-pressure is, in part at least, dependent upon the amount of salt in the food, being low on salt-free and high on diet rich in salt, though this is by no means the rule.

DISTRIBUTION OF ARTERIOSCLEROTIC LESIONS.

As regards the distribution of arteriosclerotic lesions and its relations to etiology, Harlow Brooks has given the following statistical summary based upon notes of autopsies on 400 cases.

Artery.	Cases	Etiological factors.
	400	Alcohol 149, among laborers 118, nephritis 51, syphilis 38, old age 38. Males 275, females 125.
Aorta	301	
Visceral trunks	368	
Coronary arteries	270	Alcohol 107, nephritis 33, syphilis 27, excessive tobacco 9.
Brain	132	Alcohol 48, nephritis 21, syphilis 10
Renal	81	Alcohol 13, nephritis 10 syphilis 10.
Pancreas	74	Alcohol 19, syphilis 9, senility 9
Hepatic	43	Alcohol 12, nephritis 8, syphilis 6, senility 3.
Splenic	33	Alcohol 9, syphilis 7 nephritis 4, endocarditis 2, senility 2, tuberculosis 2
Lungs		Syphilis 5, senility 5, alcohol 4, tuberculosis 4, nephritis 2
Coeliac axis and branches	19	Most of them with alcoholism. Sclerosis of mesenteric all cases with adiposis
Spinal vessels	20	Alcoholic 4 syphilitic 4, most of the rest in primary spinal diseases

ARTERIOSCLEROSIS IN THE YOUNG.

Arteriosclerosis in infants, children, and young persons while rare is not extremely so.

According to Fremont Smith, who has given an excellent review of the subject, congenital syphilis is the cause in about forty per cent of the cases, and diphtheria, scarlet fever, and typhoid fever, as well as infections in the mother during pregnancy, are important factors. The blood-pressure is not usually elevated often being as low as 70 mm.

Fig. The writer has seen one case of a boy aged six suffering from acute nephritis, complicated by lobar pneumonia, large bacillus coli abscess of the buttocks, cystitis caused by the same germ, who in spite of continuously low blood-pressure developed tortuous and apparently thickened temporal and thickened radial arteries. After a few months these arteries were no longer palpable. It is possible that these changes may have been merely mononuclear infiltration about the vessels of the adventitia.

CLINICAL MANIFESTATIONS OF ARTERIOSCLEROSIS.

Clinically, the symptoms due to arteriosclerosis usually express themselves in several groups dependent upon the arteries most affected.

(1) Cardiac, associated with myocarditis and coronary sclerosis; often with renal symptoms (see Chapter IX). As shown by Fleisher and Loeb, the myocarditis may be produced by the same cause and may be more severe than the arteriosclerosis itself.

(2) Simple coronary sclerosis, paroxysmal dyspnœa, angina pectoris, Adams-Stokes syndrome, paroxysmal tachycardia, sudden death.

(3) Cerebral symptoms.

(4) Aneurism.

(5) Intermittent claudication.

(6) Vasomotor crises (Pal)

(*a*) Abdominal pain from vasoconstriction;

(*b*) Raynaud's disease;

(*c*) Pain down arms and legs.

The clinical characteristics of the cardiac and renal cases have been discussed in Chapter IX under the head of the myocarditis which invariably accompanies them. They may be briefly summarized as shortness of breath, especially on exertion, often asthmatic or paroxysmal in character; palpitation; weakness, occasionally a considerable degree of nervousness, loss of memory, and insomnia. In advanced cases with some sclerosis of cerebral arteries there may be more or less transient irrationality, especially at night or on awakening. There may be pains over the precordium, in the shoulders, or down the arms, or in the abdomen or legs, which may be definitely associated with periods of high blood-pressure (the vasomotor crises of Pal); there may be sudden pain and sudden paralysis of a leg, disappearing on rest, reappearing after a few steps are taken (intermittent claudication, Charcot, Erb); or there may be severe precordial pain with a feeling of weight and constriction over the sternum and an utterable fear of impending death (angina pectoris). On the other hand, the hand or foot may become cold or numb, the pulsation disappear from the arteries, intense pain set in (Raynaud's disease), or finally be followed by gangrene (thromboangiitis obliterans). Still further the patient may suffer from all the signs and symptoms of aneurism.

On physical examination the radial arteries may or may not be found to be thickened or beaded (atheromatous), dependent partly upon the distribution of the sclerosis, since the radial artery may be spared. Some writers state, however, that in men who do hard manual labor the radial arteries are the first attacked, while in those who lead a sedentary life sclerosis may appear very early about the base of the aorta, and the radial, nevertheless, may be perfectly normal.

The artery in which the sclerosis is next most readily observed is the temporal, which usually stands out like a cord or is very tortuous, and when pressed against the bone feels thickened and leathery. This tortuosity may also be present in the brachials and even in the abdominal aorta, and is probably brought about by the stress of the arterial tension exerted upon the walls, which are in some places weaker and less elastic than in others so that we have a force (blood-pressure) which is exerted equally on all sides against walls which interpose a greater resistance on one side than on the other, hence the curvature results. As might be expected, the tortuousness is therefore greater when the disturbing force is high (high blood-pressure) and less when it is low, as shown in the figure (Fig. 157).

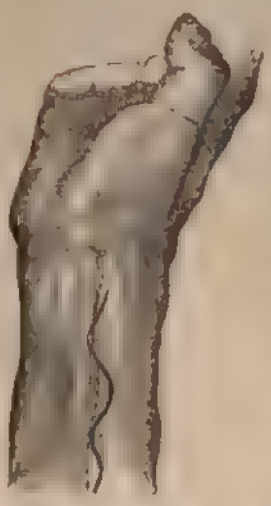

Fig. 157.—Tortuous radial artery. (After Pal.) Showing course of the radial artery at 300 mm. Hg blood-pressure. Broken line, course of the artery at 85 c.m. blood pressure, after amyl nitrite.

Other superficial arteries which may be felt are the brachials, axillaries, facials, popliteals, and dorsalis pedis.

Changes in the Retinal Vessels.—Hirschberg in 1882 called attention to the fact that changes in the retinal vessels constitute an early sign of arteriosclerosis, and later demonstrated that this change was normal in old persons and usually began in the fifth decade. Friedenwald and Preston examined twenty-three persons suffering from general arteriosclerosis, and found only seven normal retinas among them.

De Schweinitz gives the following criteria for sclerosis of the retinal vessels:

(1) **Suggestive Signs.**—Uneven caliber and undue tortuousness of the retinal arteries (corkscrew form), increased distinctness of the central light streak, an unusually light color of the artery, and alterations in the course and caliber of the veins.

(2) **Pathognomonic Signs.**—Changes in size and breadth of the arteries, loss of translucency, lesions in the arterial walls consisting of white stripes in the form of perivasculitis, indentation of the veins by the stiffened arteries, tortuousness of veins and white stripes or varicosities along their courses, œdema of the retina in the form of gray opacity around the disk or following the course of the vessels, hemorrhages as linear extravasations or roundish infiltrations. Sometimes very sudden changes in the caliber of the retinal arteries may be seen accompanying vasomotor crises.

X-ray Examination.—Absolute proof of arteriosclerosis is also given by the X-ray, by which calcified plaques along the course of deeply situated arteries (popliteals, femorals, abdominal aorta, etc.) may be discerned as distinct shadows ranged along the course of the artery. These may be brought out more distinctly by using two stereoscopic pictures instead of one. Unfortunately, it has not been possible to discern sclerosis of the coronary arteries in this way.

Sclerosis of the Abdominal Aorta.—Arteriosclerosis of the abdominal aorta and splanchnic vessels is very common, as has been shown by Hasenfeld, Bond, Brooks, Ortner, and Gilbride. In fact, it may almost be diagnosed with certainty when the blood-pressure is elevated. Occasionally the course of the abdominal aorta may be felt to be tortuous. Sclerosis of the abdom-

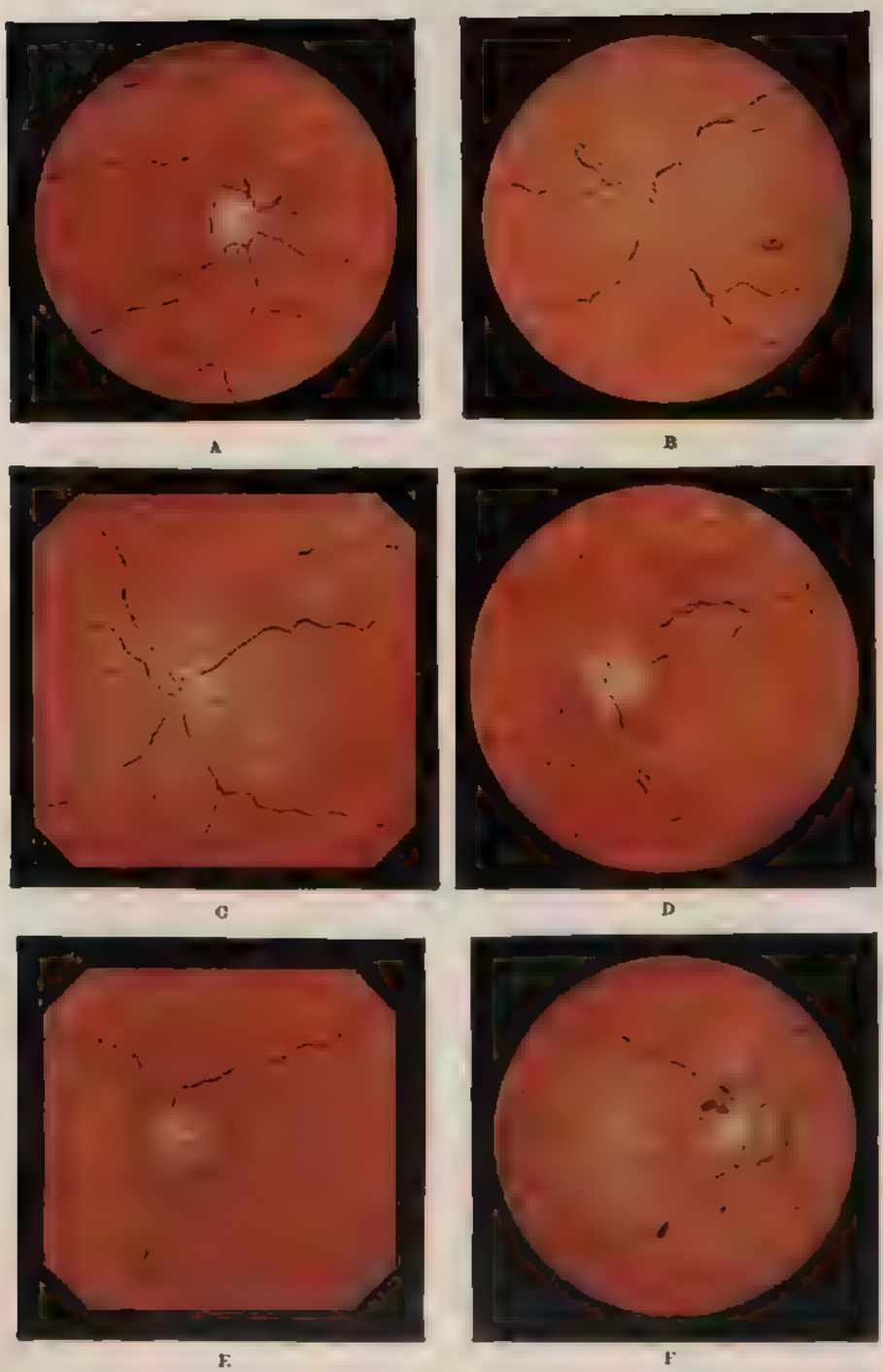

FIG. 158.—Retinal changes in arteriosclerosis. A, Normal fundus. B to F, successive changes occurring in arteriosclerosis, including palpid arteries (B), later assuming a silver-wire appearance (C), indented veins (B, C) afterward showing sacculiform enlargements (D, E), corkscrew capillaries (C, D); corkscrew arteries and veins (D, E), perivasculitis (C, D), sclerosis of vessels (F), œdema of disk (B, C, D, E), hemorrhages (C, F).—D. (After de Schweinitz.)

inal vessels is not infrequently accompanied by crises of severe abdominal pain not unlike those of tabes (abdominal vasomotor crises), but these may also be present from simple pulsation of the abdominal aorta when tugging upon loose peritoneal moorings. Sclerosis of the pancreatic artery is often accompanied by diabetes mellitus.

BLOOD-PRESSURE AND PULSE.

In arteriosclerosis the mechanical factors affecting blood-pressure tend to approach those in a system of rigid tubes,—a high pressure throughout systole, a low pressure in diastole. In such a system we should have, as a rule, a greater difference between pressure in systole and in diastole than when the normal elasticity tends to keep up the diastolic pressure, so that the pulse-pressure is often more than 50 to 60 mm. rather than being nearer 30 or 40 mm. as in the normal individual.

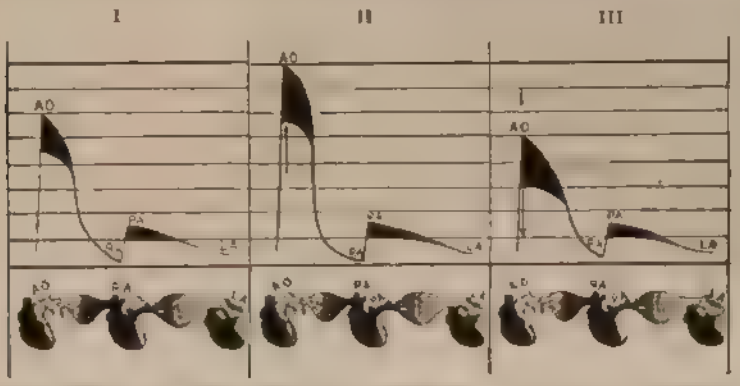

FIG. 159.—Effect of arteriosclerosis upon the circulation. I, normal. II, arteriosclerosis, with high peripheral resistance and anacrotic form of pulse wave; the arrow points to a rise in maximal and minimal pressure and increased pulse-pressure. III, arteriosclerosis with low peripheral resistance, showing low blood-pressure and increased pulse-pressure and collapsing pulse.

Pulse. The pulse may assume any form whatever, from collapsing and almost water-hammer in character to an anacrotic plateau, or even in rare cases to a pulsus tardus. These depend upon the relation between strength and size of beat and outflow through the arterioles. Thus, if the peripheral arteries or any large areas of blood-channels are dilated and lacking in elasticity, there will be a momentary rise in pressure at the beginning until the pressure wave is transmitted from the aorta to the periphery. When it reaches this point there is a sudden outflow through those vessels and a sudden fall or collapse, which is greater than it would be in a more elastic system (see Fig. 159). On the other hand, if the peripheral outflow is small, the pressure in the non-elastic system quickly rises higher than in an elastic system and remains so throughout systole, forming a systolic plateau (anacrotic pulse) with a large rapid rise and plateau reaching to the end of systole, then a gradual fall during diastole. The pulse form accordingly gives us the information in arteriosclerosis as in other conditions (see page 44), namely, indicates low peripheral resistance when it is collapsing and high peripheral resistance when it is anacrotic or sus-

tained. The pulse may either be quite large or very small, dependent upon the degree either of vasoconstriction or of endarteritis. Its character may be very variable; it may be quite quick and collapsing, corresponding to a general rigidity of the whole vascular system, or the vessel may fill rapidly, remain well sustained with long systolic plateau, and may then decline either rapidly or slowly. However, the lumen of the radial artery may have decreased so much from an endarteritis that the filling of the artery is slow and the up-stroke on the pulse-tracing very oblique, just as would be typical of aortic stenosis. This is not extremely common, and the very quick up-stroke is the form most frequently seen. On the other hand, in rarer cases when, as Romberg and also Hasenfeld have pointed out, the splanchnic vessels are not involved, the maximal blood-pressure may be quite normal (110–120 mm.) and the minimal also (90 mm.).

Blood-pressure. — The blood-pressure is often high. Thayer found in his studies of post-typhoid arteriosclerosis that the maximal blood-pressure was usually 20–30 mm. higher than for normal individuals of corresponding age.

Romberg and Sawada, on the other hand, found that this occurred in only 12.5 per cent. of all arteriosclerotics, while Groedel found hypertension in only 37 per cent. of 446 cases of arteriosclerosis free from chronic nephritis. Dunin found similar results. Israel, however, found hypertension (over 140 mm. Hg or 180 cm. H_2O by Recklinghausen apparatus) in 64.4 per cent. of 45 cases of arteriosclerosis. The minimal pressure was also increased, but less than the maximal. Israel gives the following average figures:

	Max.	Min.	Mean	Pulse pressure simplicidade
Normal				
cm H_2O	170	110	140	60
mm Hg	125	81	103	44
Arteriosclerosis—				
cm H_2O	240	140	190	100
mm Hg	177	103	140	74
Average increase				
mm Hg	52	23	37	30

Israel's figures accord well with the writer's experience (using the Erlanger apparatus). The highest of these blood-pressures are seen in cases with chronic nephritis (Israel, Janeway, Horner). The writer has often found a maximal pressure of 220 mm. Hg with a minimal of 160, though usually in association with nephritis.

As has been seen under cardiac overstrain, the presence of arteriosclerosis has a marked effect in impairing the bodily strength and the ability to withstand strain. The diminution in arterial bed increases the total work of the heart, and the patches of arterial fibrosis prevent the arteries from dilating under functional activity. On the other hand, the loss of arterial elasticity removes a factor which tends to propel the blood during diastole and thus to maintain the blood flow at the least expenditure of energy by the heart. As a result of this factor, the heart is compelled to increase its systolic output (increased pulse-pressure) under normal condi-

tions and hence has little ability for further increase in reserve. Muscular effort therefore gives rise to signs of greater strain than in normal individuals, greater increase in blood-pressure, and greater fatigue.

The intensity of vasomotor reactions varies considerably in different cases of arteriosclerosis. In some cases, as Romberg has shown, the vasomotor reaction of the arm vessels to cold may entirely disappear, while in others (vasomotor crises) the reactions are so intense as to produce ischæmia of the parts.

The Second Aortic Sound.—Corresponding to the high blood-pressure there is also accentuation of the second aortic sound, which on the one hand may be due to the heightened blood-pressure and the greater tension of the aortic valves, or, on the other, to the thickening and partial calcification of the valves themselves, which gives rise to a louder sound than usual when the valves strike together, even under the usual pressure. A marked accentuation of the aortic second sound therefore always leads to the suspicion of arteriosclerosis, even in the absence of thickening in the walls of the superficial vessels. However, it is not pathognomonic, since it may often be heard in cases where no special sclerosis is present, especially at times when the heart is acting strongly and probably giving forth a larger output into the aorta at each systole, as in typhoids with dicrotic pulse or in perfectly healthy young persons during attacks of palpitation. In such cases the accentuation of the second sound is t r a n s i t o r y.

BLOOD COUNT IN ARTERIOSCLEROSIS.

The blood count may vary considerably, first on account of the great variety of diseases associated with arteriosclerosis, and secondly, because the latter is sometimes accompanied by polycythæmia or erythræmia

There are no blood changes which in themselves can be said to be definitely associated with arteriosclerosis.

AORTIC SCLEROSIS.

When the aortitis near the base of the aorta is marked, and especially if calcified plaques are present, the first sound as well as the second may be changed and may be accompanied by a loud murmur which is usually transmitted to the carotid and brachial arteries, resembling that heard in aortic stenosis but less intense. Since the condition is much more common than the latter, this murmur is also more commonly due to this cause, but in the absence of the characteristic pulse it is quite indistinguishable from that of aortic stenosis, for both arise at the same site at the same time and are transmitted in the same way The murmur is often accompanied by a marked thrill having the same distribution and is followed by a distinct diastolic shock.

As regards sclerosis of the aorta alone, Bittorf has found that it frequently occurs at an average age of fifty-five in syphilitics as a result of the usual factors, sometimes a single trauma to the chest may seem to be the important element in the etiology. It is especially common in syphilitics and fat persons, and is frequently associated with pale ashy-gray color, very high blood-pressure 170 to 220 mm., occasionally difference in size of the pupils, pains over the chest and down the arms, œdema over the sternum,

unilateral dilatation of veins in second and third interspaces, ringing aortic second sound without diastolic murmur, hypertrophy of the heart, often pulsus celer, rarely pulsus tardus or pulsus paradoxus. Cardiac pain may be present, often felt just after percussion, and described as something boring through the sternum sometimes with a feeling of constriction, sometimes radiating to the arms and neck. Occasionally spells of weakness in the arms may be felt not unlike intermittent claudication.

The differential diagnosis from aortic stenosis is made by the gradual upstroke on the pulse tracing in the latter case as contrasted with the sudden upstroke and plateau in the former, from aortic insufficiency by the diastolic murmur and high pulse-pressure; from aneurism by the percussion and fluoroscopic findings. Nevertheless, it must be admitted that many doubtful cases arise.

SCLEROSIS OF THE PULMONARY ARTERY.

Primary sclerosis of the pulmonary artery is not extremely rare, but is difficult or impossible to diagnose with certainty, but the presence of very loud sounds in the pulmonic area or of a rough systolic murmur heard loudest at the pulmonic and transmitted upward towards the left clavicle arouses the suspicion of a pulmonary sclerosis, especially if signs of congenital lesion are absent and the murmur is not heard over the carotid. However, sclerosis of the pulmonary artery is often secondary to mitral stenosis and emphysema.

Case of Primary Pulmonary Sclerosis.

Romberg reports the case of a man, aged 24, who had had no infectious diseases except measles as a child and a recent slight muscular rheumatism, three months after which he began to have gradually increasing shortness of breath, epigastric pressure, occasional headaches and giddiness, and his color became very blue. On examination he showed marked cyanosis over the face, body, and limbs. There was a pulsation due to the right ventricle in the fourth interspace 4 cm. inside the mammillary line and thence inward to the sternum; also a smaller pulsation (left ventricle) in the fifth interspace mammillary line, cardiac dulness 7 cm. to right, 15 cm. to left. Both pulmonic sounds were louder than the aortic. Pulse small, regular, 116. Liver enlarged; spleen enlarged. No œdema, no swelling of vessels of neck.

Probable diagnosis (Curschmann), congenital heart lesion. Patient gradually became worse; digitalis was without effect. Died one month after admission.

Autopsy showed enlarged heart, right ventricle hypertrophied and forms the entire apex, and the conus arteriosus and right auricle are especially hypertrophied. All the valves intact and normal; aorta free from sclerosis, but unusually small. Ductus arteriosus closed. Tremendous sclerosis and atheroma of the pulmonary artery and all its branches.[1]

The murmur may be distinguished over the abdominal aorta and the femoral artery, though the thrill is rarely transmitted so far. There is no Durozicz double murmur unless aortic insufficiency is also present.

Sanders has recently collected similar cases from the literature.

TREATMENT.

The general treatment of arteriosclerosis is mainly prophylactic, hygienic, and dietetic, and actual specific treatment is of far less value.

Diet. — Carefully selected diet is a most important factor, restriction being in both quality and quantity. The general diet given in heart cases (see page 167) is of great benefit here, or equivalent diets with

[1] Notes of a case of pulmonary arteriosclerosis (O. A. K.) secondary to mitral stenosis are given on p. 354.

this as a basis. However, in simple arteriosclerosis the quantity taken at a time need not be so greatly restricted, but the total quantity in twenty-four hours should not exceed twenty-five hundred calories, and should always be near the lower level for proteids, and as free as possible of purin bodies (nitrogenous extractives such as are found in meat), creatinin, etc., and also of salt. The more recent studies quoted above seem to indicate that excess in salt is almost as injurious as are excesses in alcohol, and that the salt mackerel of Boston is as dangerous as the beer of Milwaukee. For the sclerotic danger probably lurks in the Smithfield ham or the cold smoked tongue as well as in the Baltimore rye or the Martini cocktail (Beyer, Barié, Hadfield). The patient's safety lies in milk, eggs, potatoes, bread, other carbohydrates, butter, and the simpler fruits.

Restriction of Liquids. — On the other hand, the liquid intake also should not be excessive, since drinking large amounts either of water or of beer seems to favor sclerosis (Krehl), but the amount ingested should remain in the vicinity of fifteen hundred cubic centimetres a day, some persons thriving best at five hundred cubic centimetres above, some at five hundred cubic centimetres below this level.

Tobacco and alcohol should be dispensed with entirely if possible; if the patient insists on taking small quantities, one or two light dry cigars, as thin as possible (Lee), or "stogies," a day are perhaps the mildest that one may prescribe. Cigarette smoke is usually inhaled and pipes are very heavy. Thick Havana cigars should be entirely prohibited.

As to alcohol, if the patient insists upon taking a small quantity, this should be limited to an occasional glass of claret or white wine, or perhaps a single glass of beer at rare intervals. The latter in large quantities is especially undesirable, both on account of the large amounts of liquid taken and because it contains both alcohol and proteid and purin substances extracted from the yeast. Gin is perhaps more dangerous than whiskey.

Coffee and tea should be taken in only small quantities, since the vasoconstrictor action of the caffeine favors the onset of spasmodic vasoconstriction (vasomotor crises), and, on the other hand, the increase of blood-pressure itself brought on by caffeine is damaging to the arteries. However, it must be stated that, in contrast to nicotine, lead, adrenalin, etc., injections of caffeine into animals have thus far failed to bring on arteriosclerosis and that perhaps the deleterious effect of caffeine may be overestimated.

Hydrotherapy. — Systematic hydrotherapy is of considerable value in arteriosclerosis, especially the use of warm baths, warm douches (Brieger), or alternating warm and cold douches (Riley) applied both locally and generally. They owe their efficacy to the vasodilatation which they bring about, and hence must be classed in effect with the drugs of the nitrite group. In most cases the effect of a good warm douche or warm bath is more marked and more lasting than that of any of these drugs, and it is further devoid of that certain residuum of deleterious effect which all drugs leave behind them. So that, while one cannot agree with Brieger that arteriosclerosis can be entirely cured symptomatically by proper hydrotherapy, nevertheless warm baths and warm showers

once or twice a day should be an indispensable part of the treatment of every arteriosclerotic. Cold baths should be avoided, since they precipitate vasomotor reactions, which in the arteriosclerotic may amount to vasoconstrictor spasm.

Drugs. — **Potassium Iodide.** — As to drugs, universal experience points to the efficacy of potassium iodide in doses ascending from 0.3 Gm. (gr. v) t.i.d., p.c., to as high as 4 Gm. (℥i); some clinicians favoring the smaller, some the larger doses. In the writer's experience doses under 1 Gm. (gr. xv) seem to have some effect in alleviating symptoms, and when there is a suspicion of lues the dose should be increased still further. (The therapeutic action and its limitations are discussed in Chapter V.)

It has been attempted to settle the question experimentally by determining the effect of potassium iodide upon the course of adrenalin atheroma in rabbits. Koranyi, Boveri, and Cummins and Stout, who were the first to undertake these investigations, all reported that potassium iodide or iodipin, when injected during the time that adrenalin was being injected, inhibited the production of atheroma. However, it must be borne in mind that Biland, Loeb and Githens, Adler and Hensel found that large doses of potassium iodide seemed to increase rather than inhibit the atheromatous changes. It is at present impossible to tell exactly what quantity represents the optimum dose for human beings, and whether therapeutic doses ever reach the stage of harmfulness.

While sclerosis of the pulmonary artery secondary to the pulmonary stasis of mitral stenosis is relatively common, primary sclerosis of this artery is rather rare.

Nitrites. Next to the iodides in general use is the group of nitrites, — amyl nitrite, nitroglycerin, sodium nitrite, erythrol tetranitrate. These drugs are of value for symptomatic treatment, to relieve pain or discomfort for the time being, but they exercise no inhibitory influences upon the progress of the arteriosclerosis, as has been shown for adrenalin arteriosclerosis. On the other hand, their effect upon the symptoms due to arteriosclerosis, the pain of intermittent claudication, of angina pectoris, of the abdominal and peripheral vascular crises, is most remarkable, and in this regard they are invaluable (Lauder Brunton). However, in their administration it must be borne in mind that persons with arteriosclerosis seem to have considerable tolerance for nitrites (page 188), and to bring about vasodilatation and fall in blood-pressure much larger doses must be given than is necessary to produce the effect in normal individuals. Accordingly, as indicated in Chapter V, the drug should be administered in increasing doses until the physiological effect (flushing, throbbing in head, ringing of the ears) is obtained, and then continued in a dose just a little smaller than this. One need not be surprised, however, to find that this dose for a person with arteriosclerosis, particularly a colored person, may be ten or even twenty times the average dose for a normal individual. When such is the case the blood-pressure is probably a beneficial compensatory phenomenon, and the nitrites should be discontinued.

In the chronic hypertension of arteriosclerosis venesection is not only useless but often harmful.

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XI.

VASOMOTOR CRISES AND THE ANGIONEUROTIC LESIONS.

VASOMOTOR CRISES

GENERAL CONSIDERATIONS.

The general clinical manifestations of arteriosclerosis bear a close relation to the condition described by Pal as "vasomotor crises," under which he includes all conditions which are associated with more or less sudden constriction or dilatation of the arteries, and whose symptoms and signs disappear or markedly diminish as soon as this paroxysmal change in the blood-vessels passes off. There are accordingly

(1) Vasoconstrictor crises, usually associated with hypertension.

(2) Vasodilator (hypotension) crises.

The vasoconstrictor crises Pal divided into

(1) Abdominal type. (2) Pectoral type. (3) Cerebral type. (4) Crises in the extremities. (5) Crises in the large arteries.

The vasodilator crises according to Pal include

(1) Ordinary syncope. (2) Surgical shock. (3) Collapse after infectious disease or most poisonings. (4) Erythromelalgia and many other "trophic" skin disease. 5) Occasional cases of tabes with lancinating pains and low blood-pressure. (6) Various attacks of weakness in Addison's disease.

Probably no unit cause exists for the crises themselves: the visceral crises and lancinating pain in tabes, the painter's colic, the uræmic convulsion, the delirium of the cerebral sclerotic, the pain of angina pectoris, and the attack of cardiac asthma seem to have little etiology in common except their relation to the sympathetic nerves. However, all manifest high blood-pressure, and, according to Pal, all are relieved by artificial depression of blood-pressure. It is, therefore, not unlikely that however diverse the ultimate causes of the condition, the cause of the symptoms is high blood-pressure with localized vasoconstriction. The variation in the areas of constriction in regions whose arteries are already sclerotic accounts for the occurrence of the different symptom complexes.

As to treatment, the statements of Pal would lead one to believe that they are all relieved by vasodilators, especially nitroglycerin and the nitrites, occasionally by sodium thiocyanate, and that marked improvement results while the blood-pressure is lowered. The symptoms return if the blood-pressure again rises. (Pal, also Hertz, and Norrero.) However, Prof. Barker's experience at the Johns Hopkins Hospital does not warrant such sweeping conclusions.

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[1] Buerger's studies indicate that there are organic lesions in some of these cases.

Case of Abdominal Vasomotor Crises (Quoted from Pal.)

P. V., sausage maker, aged 57, had rheumatism 14 years ago, and for the past year pain and pressure in the epigastrium, especially on taking a deep breath. Has occasional paroxysms of extreme dyspnœa and palpitation of the heart, but always has some shortness of breath. He was formerly a heavy drinker, now drinks two or three litres of beer a day as well as a half litre of wine and some whiskey! He also smokes in moderation.

On admission April 7, 1904 he was found to be a well-nourished man, slightly cyanotic. Lungs clear, respiration 34. Heart. Maximum impulse in sixth interspace two fingers' breadth beyond mammillary line. Dulness extends to third rib above and two fingers' breadth beyond the right margin of the sternum. Sounds quite clear at apex and base, second aortic sound not accentuated. Pulse 68, radial walls stiff, blood pressure 225. Liver enlarged, spleen not palpable. Slight œdema of feet and legs. Urine 2000 c.c., sp. gr. 1010, albumin 1.5 Gm. per litre.

Patient was given 0.5 Gm. (gr. viii) sodium thocyanate t.i.d. to diminish his blood-pressure.

April 21. Patient delirious; blood-pressure 110. Thocyanate discontinued, whereupon delirium disappears. The chart in Fig. 160 shows the course of the blood pressure, pulse-rate, and respiration. The patient was free from other exceptional symptoms from April 7 to May 1. May 1, 8.00 11.30 A.M. Feels hot and cold. 11.30. Sudden attack of severe pain and great feeling of pressure in epigastrium. 11.35. Pains in back and third to seventh vertebræ. Cries out with pain, and also cries "I am choking." Lungs clear. Cardiac dulness only to right sternal margin and to two fingers' breadth within left mammillary line. 11.40. Symptoms diminish but pressure in epigastrium still present. 11.41. Symptoms reappear. 11.42. A second severe attack as before. 11.55. Diminution of symptoms, pains less. After a few minutes patient has a third momentary attack with blood-pressure over 200 mm. Hg, which then subsides. 12.10. Feels better. 12.55. Still better. Free from attacks until May 3, during which time he receives 0.5 Gm. (gr. viii) duretin t.i.d. On May 18, sodium thocyanate was again given, which lowered blood-pressure but caused delirium.

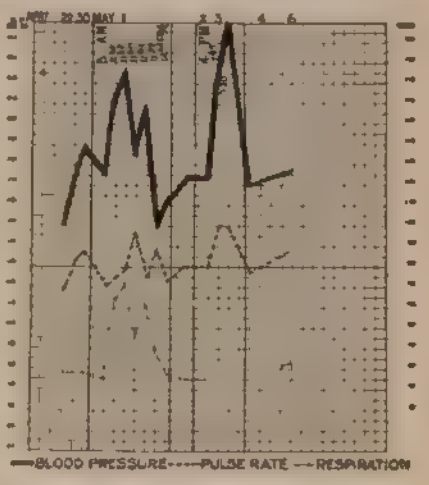

Fig. 160. Blood-pressure chart of P. V. Typical vasomotor crisis.

From that time until discharged frequent attacks of pain and hypertension.

Pal reports similar hypertensive crises in association with the colic of lead poisoning and also with the visceral crises of tabes,[1] the pain being always relieved when the blood-pressure is brought down by amyl nitrite or nitroglycerin, as, for example, in the following case.

Case of Abdominal Crisis in Lead Poisoning

N. J., painter, aged 31, has had lead colic twice before. Was free from it on change of occupation but it returned when he again worked in lead. Drinks little; denies lues. He has had abdominal pain for three weeks. During past few days has

[1] The claim of Pal that a similar association of pain with high blood-pressure exists with the lancinating pains of limbs cannot be maintained since the pains in his own cases are sometimes associated with hypotension, sometimes with hypertension.

had continuous cramps, loss of appetite and no stool. He is pale and has a marked lead line. Pupils react readily. Lungs clear. Heart normal; pulse rather hard. Abdominal walls tense, tender on both sides; spleen just palpable.

July 23	8.30 P.M.	B. P. 130	Slight pain
	9.1 P.M.	P. 168	B. P. 170. Increased pain
	9.13 P.M.		Amyl nitrite inhalation
	9.15 P.M.	B. P. 105	No pain
	9.17 P.M.	B. P. 165	Pain again lasting then over one-half hour with same B. P.
	9.45 P.M.	B. P. 95	After amyl nitrite which again gave relief
	9.48 P.M.	B. P. 140	Pains return
	12 M.	160	Further increase of pain
	4.20 A.M.	B. P. 135	Pains diminish under amyl nitrite. They return again but
	6.30 A.M.	B. P. 85	Pains disappear under amyl nitrite

After July 25 blood-pressure was always under 130 during last four days under 110), the patient was free from pain and bowels were regular.

Case Illustrating the Cerebral Crisis

The following case, illustrating what Pal terms the cerebral type of vascular crisis was under the writer's care at the Johns Hopkins Hospital.

J. M. C., grocer, aged 52, who had suffered repeatedly with myocarditis, hypertrophied heart, irregular pulse, and general anasarca, entered the Johns Hopkins Hospital in September, 1903.

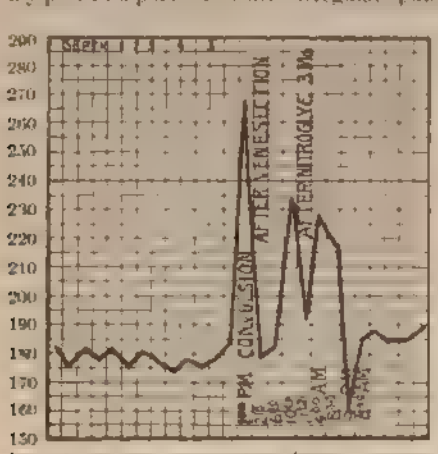

Fig. 161.—Blood-pressure chart showing a vascular crisis of the cerebral type.

Oct. 24. Restless at night. Left pupil larger than right. Both react normally.

Nov. 2. Very weak. Pulse weak and irregular. Liver enlarged.

Nov. 5. At 12.30 P.M. began to complain of general discomfort with numbness in legs; complained of nervousness and restlessness.

At 12.45 P.M. the restlessness became very marked; he began to strike out with his hands and to try to get out of bed. Was at this time conscious and able to understand questions. (Blood-pressure curve shown in Fig. 161.) No aphasia. Pupils equal and dilated. Head and eyes drawn to right and rigid. There was some twitching of muscles of both arms and hands. Reflexes of right arm slightly exaggerated. Soon became cyanotic and vessels of neck stood out. Became unconscious. Respiration stertorous. Blood-pressure 270 mm. Hg. After 600 c.c. of blood had been withdrawn from left arm cyanosis slowly subsided, respiration becoming less stertorous and blood-pressure falling to 180 mm. Hg.

Becomes conscious after catheterization at 7.30 P.M. Still picking at bedclothes, which continued until next morning. He was then mentally clear by 11 A.M. and pupils reacted to light.

Nov. 6. 9 P.M. Remained clear and recalled hallucinations of previous night, realizing them as hallucinations. Blood-pressure 160.

Had no further attacks of this kind and blood-pressure remained below 190.

Died March 28, 1904. Autopsy showed chronic myocarditis; heart 1000 Gm.; chronic adhesive pericarditis, coronary sclerosis, hydronephrosis, and stone in right kidney.[1]

[1] It is possible that this attack may have been due to transitory cerebral oedema like that described by H. Cushing and James Bordley, Subtemporal Decompression in a Case of Chronic Nephritis with Uraemia, with Especial Consideration of the Neuroretinal Lesion, Am. J. M. Sc., 1908, cxxxvi, 484.

INTERMITTENT CLAUDICATION.

This condition is always associated with sclerosis of the femoral, popliteal, or one of the other arteries of the leg which are usually pipe-stem in character. Often the atheromatous changes are readily demonstrable by the X-ray. Owing to the narrowed lumen of the artery, the amount of blood that can flow through it is limited, but this is sufficient to supply the muscle when at rest. During slow walking the CO_2 produced by the muscle and the oxygen needed by it increase greatly. If the arterial flow is sufficient, no symptoms appear; but when rapid walking or running is begun, there is a sudden increase in the oxidation in the muscle, and, since the blood supply cannot keep pace with it, asphyxia of both the muscle and its nerve endings sets in, accompanied by paralysis of the limb and often intense pain arising from stimulation of the sensory fibres by the CO_2. The patient is compelled to halt. During the rest the CO_2 production falls, and the slow circulation is able to carry off the excess and to supply fresh oxygen to the tissues. With the renewed aeration, function returns. The patient is able to walk again until local asphyxia sets in, and, since this will be brought about by the same amount of CO_2 as before, his walking will be limited to the same distance. He must travel in stages. (Fig. 162.)

FIG. 162. Diagram to illustrate the elimination of CO_2 by the blood in normal and sclerotic arteries. Solid line indicates CO_2 formation during halts and while walking. Broken line represents the rapidity of CO_2 elimination. * indicates the degree of CO_2 accumulation at which pain sensations set in.

CASE OF INTERMITTENT CLAUDICATION.

H. E., carpenter, aged 74, complains of pain in right foot, drinks beer and whiskey in moderation, smokes very little, and has always been healthy. In October toe was red and ached. For the past ten or fifteen years patient has been attacked by severe pains in both feet, causing him to stop in his walks. Knees never gave way. The attacks came on oftenest during exercise. On examination, thorax is emphysematous, heart slightly enlarged to left. Blowing systolic murmur heard over the tricuspid area, becoming musical over the apex, well heard in the axilla, but faint and blowing in the pulmonary area, where the second sound is accentuated. Pulse slightly irregular. Right radial more sclerotic than left. Blood-pressure 165 mm. General reddening from tarsometatarsal joints to the toes of right foot, where pulsation of dorsalis pedis is not felt. Both tibials are palpable but pulsation is well felt. Left foot normal, artery pulsating well. Both popliteals are very sclerotic.

Given nitroglycerin mg. 1 (gr. $\frac{1}{100}$) t.i.d., alternating with sodium nitrite 0.2 (m. iij) t.i.d. He was somewhat improved by treatment, but left the hospital a few days later.

Prognosis.—Since the claudication is simply part of the general arteriosclerosis, the prognosis is bad, for the coronary arteries, aorta, and cerebral arteries may be involved. Sometimes, however, the arterial change is confined to the limbs, occurring simply as degeneration of the media with atheroma, exactly as is found in experimental adrenalin arteriosclerosis. In that case the prognosis as to life is, of course, better.

HYPOTENSIVE VASOMOTOR CRISES.

The so-called "hypotensive" crises seem to bear no relation to arteriosclerosis, but rather to trauma, action of toxic substances, and perhaps to cutaneous diseases. They are in the main associated with depression of the vasomotor system and have been discussed elsewhere. The one condition with paroxysmal depression of the blood-pressure which may owe its origin to arteriosclerosis is paroxysmal tachycardia (see page 560).

ANGEIONEUROSES

Maurice Raynaud in 1862 described many cases of this group, especially of the condition which bears his name. He showed that the three phenomena manifested in these conditions are:

1. Local syncope, *i.e.*, blanching from absence or diminution of blood in the arteries of the part affected: Raynaud's disease a spasmodic vasoconstriction. This is usually symmetrical in its distribution, affecting the ends of the extremities, *i.e.*, toes, hands or feet, arms or legs. The trouble in one extremity is frequently more intense than in the other. Often it leads to formation of bullæ, ulceration, and to symmetrical gangrene (Raynaud's disease).

2. Local asphyxia, *i.e.*, presence of a venous blood, that is to say of a blood insufficiently oxygenated, causing blueness of the part (now designated as acrocyanosis) with a distribution corresponding to that of Raynaud's disease.

3. Local hyperaemia, giving rise to redness (as in the condition termed erythromelalgia by Weir Mitchell).

Later investigations have enabled Cassirer as well as Barker and Sladen to epitomize the symptoms of vasomotor disease as follows:

The vasomotor symptoms include (1) hyperæmia, (2) syncope, and (3) asphyxia, the sensory, (1) pain, (2) hyperæsthesia, (3) anæsthesia, (4) paræsthesia, the trophic, (1) ulceration, (2) gangrene, (3) dystrophies of the skin (Barker and Sladen). They affect the fingers and particularly the toes. The chief types of disease are acrocyanosis (Cassirer), erythromelalgia (Weir Mitchell), and Raynaud's disease.

The symptoms may be arranged as follows in ascending scale (Barker and Sladen):

1. Acrocyanosis.
   Vasomotor symptoms—venous stagnation and hyperæmia in fingers and toes with cyanosis, sensory and trophic disturbances absent.
2. Acroparæsthesia.
   Acrocyanosis—sensory symptoms (paræsthesia, numbness, pain, and tingling).
3. Erythromelalgia.
   Vasomotor—hyperæmia (arterial). Sensory—pain.
4. Raynaud's disease (of the symptomatic).
   Vasomotor—hyperæmia, syncope, and asphyxia. Sensory—pain, anæsthesia, paræsthesia. Trophic—gangrene and scleroderma.

As might be expected, there are many cases with symptoms intermediate between these groups and many transitions from one to the other (Sachs).

Pathology.—Raynaud realized that the gangrene in the disease which bears his name differed from ordinary gangrene and directed his first investigations to the state of the arteries. He found that, though the pulse became very small or impalpable during the attacks of blanching, it returned to normal volume between attacks. He made very careful pathological studies of the extremities in a number of cases, and finding the arteries clear concluded that the trouble was of vasomotor origin, a view which he supported by demonstrating transitory changes of caliber in the radial, popliteal, and retinal arteries, associated with the attacks. In accordance with these studies of Raynaud the vasodilation of erythromelalgia corresponds to a period of paralysis of the vasoconstrictor nerves (sympathetic paralysis) quite similar to the active hyperæmia which Claude Bernard produced in the rabbit's ear by cutting the cervical sympathetic. Just such a local paralysis of the vasomotors produced by the overheating of a hand or foot benumbed by cold gives rise to the condition of "chilblains." The latter condition is always associated with overheating after exposure to cold and often with formation of blebs, while attacks of erythromelalgia may occur spontaneously from slight emotional or nervous disturbances or from slight exposure to cold without overheating. There is rarely bleb formation. An attack of chilblains induced by overheating may thus be continued in spontaneous attacks of erythromelalgia.

Raynaud's disease, on the other hand, corresponds to an extreme vasoconstriction, like that produced in ergotism. Raynaud himself was so much impressed with this similarity that he made searching inquiries in all his cases regarding the character of rye bread taken, and conducted an extensive series of experiments upon ergotism in various animals. He was forced to discard the ergot hypothesis by the absence of any obtainable evidence of ergot ingestion, but the parallelism between the two conditions remains.

Case of Mild Raynaud's Disease.

A. S., a trained nurse, aged 30, was always healthy until the age of nineteen, when during her period of training she was compelled to have a small ovarian cyst and one ovary removed. For some years she suffered considerable pain from adhesions, so that three years ago these were broken up by a second operation. She bore the operation well, but during convalescence three weeks later had a fainting spell, since when she suffers from severe palpitation. For the past two years she has found that in cold weather both her hands and forearms become absolutely white, cold, and numb. This condition is soon relieved by rubbing or by placing them in a basin of warm water, but is sufficiently severe to prevent her from accepting a very desirable appointment in a colder climate.

Between attacks the patient seems perfectly healthy, has a good color. All the arteries are soft. They appear to be of normal caliber and pulsate normally. The heart is normal in size, but moves 7 cm. from left to right as the patient turns from one side to the other. The right kidney is also palpable and very movable. The rest of the abdomen, and the lungs are clear. The blanching of the hands occurs less frequently and less intensely when the patient's health is good, but it occurs much more frequently when the patient is excited.

Nitrites, belladonna, lignalis, bromides, and a large number of cardiac stimulants have been tried by the patient without marked effect.

THROMBOANGITIS OBLITERANS.

In recent years Weiss and v. Winiwarter, and especially L. Buerger, have discovered a group of cases in which symptoms at times simulating those of the vasomotor trophoneuroses are produced by complete occlusion of the arteries or veins with spontaneous thrombosis (thromboangitis or thrombophlebitis obliterans). In such cases the largest artery and sometimes both artery and vein become occluded by a thrombotic process of considerable extent. After a short time the fresh red thrombi within the vessels undergo organization, usually with permanent obliteration of the lumen by white fibrous tissue. There is no proliferation of new elastic fibres encroaching on the lumen as is the case in arteriosclerosis (Fig. 163), though a few elastic fibres are found in the newly formed blood-vessels.

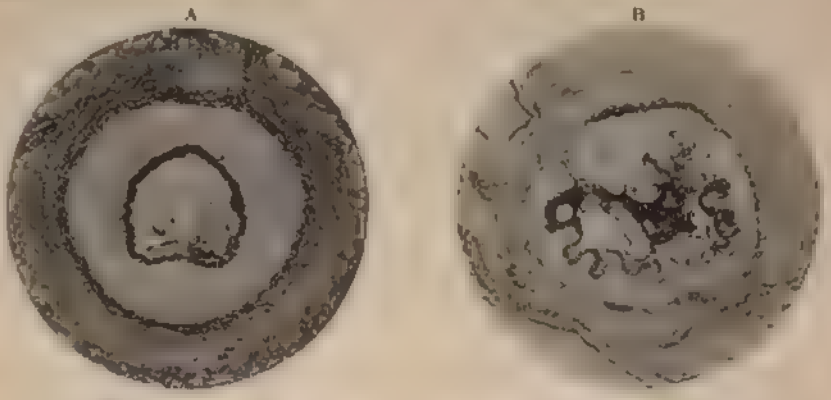

FIG. 163.—Thromboangitis obliterans (A) and endarteritis obliterans (B). (After Buerger.) The elastic fibres, stained black, are absent from the organized thrombus in A but present in large numbers in the arteriosclerotic lesion B.

This was the condition first sought for by Raynaud to explain the origin of symmetrical gangrene, and described by him under the head of senile gangrene. In Buerger's experience of over 70 cases, however, it is most frequent in Russian and Polish male Hebrews between twenty and thirty-five or forty, and hence is usually a "presenile" gangrene. In such cases the local syncope and ulceration are due to arterial occlusion. The red blush is due to compensatory capillary dilatation (termed erythromelia by Buerger, in contrast to erythromelalgia). Cyanosis of the limb occurs when the venous circulation is slowed from any cause.

The sensory disturbances found in the trophoneuroses are also found in thromboangitis obliterans.

The clinical picture produced by thromboangitis obliterans is sometimes so similar to that of Raynaud's disease (spasmodic vasoconstriction) that Buerger has found some undoubted cases of the former condition reported in the literature as cases of the latter.

Differentiation between Thromboangitis Obliterans and Angioneuroses.—Dr Buerger has informed the writer that he finds the following points useful for clinical differentiation:

1. There is always at least one vessel which remains permanently pulseless, while in Raynaud's disease the pulse soon returns to normal.

2. Intermittent claudication is present in most of the cases.

3. Usually one limb is affected a considerable time before the other, and the disease usually attacks the lower extremities.

4. There are exacerbations, but they come on and subside rather gradually and are not paroxysmal like Raynaud's disease.

5. Limbs which are red (erythromelia) or blue in the dependent position become blanched and ischaemic when elevated.

6. Migrating phlebitis is not infrequently associated with thromboangitis obliterans.

7. He has seen over 70 cases in Russian and Polish male Hebrews, but never in a female. Raynaud's disease occurs more often in females.

8. Onset is usually gradual, while it is sudden in Raynaud's disease.

9. The circulatory phenomena are for the most part not of "vasomotor" origin, but are due to occlusion of vessels. They therefore bear the stamp of permanency.

Nevertheless, Dr. Buerger has found a number of cases in which the clinical differentiation from Raynaud's disease was very difficult. Dr. Bernard Sachs, on the other hand, believes that the vasomotor neuroses manifest themselves in diseased blood-vessels as well as in healthy ones, and that the pathological diagnosis of endarteritis or thromboangitis does not exclude the clinical diagnosis of erythromelalgia or Raynaud's disease. Indeed it is readily conceivable that thrombosis should occur more readily in somewhat diseased arteries than in normal ones. Even Dr. Buerger has found some intimal changes in his cases. Vasoconstriction may also favor thrombosis. Moreover vasoconstriction, arterial disease, and the formation of agglutinative thrombi may, as is seen in ergot poisoning, all be produced by the action of a single toxic agent.

FIG. 104.—Hands and feet of a patient with thromboangitis obliterans showing gangrenous ulcers and the stumps of amputated toes. The arrows point to the gangrenous ulcers.

CASE OF THROMBOANGITIS OBLITERANS.

The following is the history of a case which, though at the time diagnosed as Raynaud's disease and manifesting many symptoms of the latter, in the light of Buerger's investigations appears to be one of thromboangitis obliterans.

B. F., tailor, aged 32, admitted April 14, 1903, complaining of sore toes and sore fingers. Had rheumatism at 12 years; otherwise well. Smokes ten cigarettes daily. In December, 1899, cold began to cause a burning sensation in big toe of right foot. In March, 1900, pus collected under the base of nail. The nail was removed, and four months later the entire toe. Wound did not heal well. After this, tingling in other toes when out of doors, never when indoors. In April, 1902, the fingers and thumb of the right hand began to tingle and become painful and a little later on those of the left hand. In January, 1903, the left big toe began to become gangrenous.

Physical examination on entrance, negative except for the extremities. Both hands are flushed, not blue, not tender, but there is some deformation of the second phalanx of the middle fingers. Right big toe missing, sloughs between third and fourth digits. Left great toe necrotic; tenderness and pain over both first metatarsals.

Patient complains of paroxysms of intense pain during the night, lasting five to ten minutes. Elevation of the limb, warm dressings, massage, were all without effect. Condition became worse in spite of hot HgCl, compresses, etc., and the left great toe had to be removed. The stump did not heal for several months. There was never pulsation in either popliteal; very little in either femoral. Patient discharged in February, 1904, unimproved.

During this time blood count: red blood-corpuscles 5,000,000–5,500,000. Hæmoglobin 100 per cent. Urine normal. Blood-pressure 100 to 130 mm. Hg. Pulse 80.

The following history represents a more typical case of thromboangitis obliterans (quoted from Buerger).

M. K., 44 years, Russian Hebrew, father of three healthy children, was admitted to Mt. Sinai Hospital on December 8, 1908. His limbs never troubled him until about a year ago, when he felt the presence of tender spots on the inner side of the right foot. Soon other hard "lumps" and "cords" appeared, some of these in the neighborhood of the ankle, others higher up on the leg. After two months these disappeared, only to recur after a very short interval. Since then he has never been absolutely free from peculiar "painful spots," and now, on admission, he still has signs of some of them. About three months after the onset of these symptoms he experienced pain in the big toe, especially on walking. This has become gradually worse, so that he has been unable to get about properly for almost two months. Of late he has often had cramps in the calf and instep of the right leg after walking for a short distance. His chief complaint, however, is the painful condition of the inner side of his right leg.

Physical examination showed evidences of circulatory disturbance in the right lower extremity. Both the dorsalis pedis artery and the posterior tibial were pulseless, although pulsation of both the femoral and posterior tibial artery could be easily detected.

Over the inner border of the right foot there is a red streak about one-half inch in length. This corresponds to a tender indurated mass which thins out and is lost as it is traced upward. A short distance below the middle of the leg the upper end of a hard cord can be palpated. This extends down behind the border of the tibia for more than two inches, is adherent to the skin, somewhat nodulated, and marks the centre of an area of hypersensitive, swollen, turgid skin. There are no trophic disturbances. *Diagnosis: thromboangitis* and *thrombophlebitis* of the *internal saphenous* and some of its tributaries.

On December 13, 1908, a portion of the thrombosed saphenous was removed for pathological examination.

On December 26, 1908, the physical examination was recorded as follows. In the horizontal position, the right foot has a light shade of red, this is most marked over the big toe and fades off towards the ankle. In the web between the third and fourth toes there is a superficial ulcer. On the inner side of the foot almost two inches from the internal malleolus there is a hard, cord-like nodule which is adherent to the skin. Behind the tibia there is the scar left after removal of a portion of the saphenous vein. The saphenous can no longer be felt.

On elevation of the foot blanching sets in rapidly and pain becomes intense. The pendent foot turns very red (marked erythromelia).

Further Course. February 15, 1909, the pain in the foot has been getting steadily worse, and the fourth toe is beginning to turn black. On the 23d of February *amputation at the knee* was done, at the request of the patient, for early gangrene of the fourth toe.

TREATMENT.

In the light of Buerger's pathological studies, treatment should be directed toward keeping up a rapid circulation through the part and diminishing the tendency to coagulate. To bring about the former the vasodilator drugs, especially the nitrites, should be freely used, but most of all the

mechanical methods of inducing arterial hyperæmia,—hot poultices, mustard foot or hand baths, or the Bier's hyperæmia by suction *in vacuo* (not Bier's stagnation hyperæmia). Exsanguination of the arm or leg with the Esmarch bandage, which has been advocated by some writers, has given but little clinical encouragement; and in the light of the recent pathological studies seems to be the worst possible procedure, since it provokes the stagnation it intends to cure, at least long enough to induce further thrombosis.

It is possible that the administration of sodium citrate by mouth, in doses sufficient to slow the coagulation time of the blood, might aid in diminishing the tendency to intravascular coagulation, but, since the fibrin-ferment is supplied on the spot from the cells of the intima, it is probable that this would not be of much avail.

To keep up arterial hyperæmia until the thromboangitis has been repaired is the only hope in therapy.

From the time of Raynaud to the present excellent results have been reported from the use of warm (but not too hot) poultices.

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XII.

SCLEROSIS OF THE CORONARY ARTERIES, AND ANGINA PECTORIS.

PHYSIOLOGY OF THE CORONARY CIRCULATION.

The coronary arteries have usually been considered to be terminal arteries in the sense of Cohnheim, that is, that their branches did not anastomose with one another sufficiently to maintain an adequate circulation, and infarction follows their occlusion. This is correct under most clinical conditions, and Porter has found experimentally that the infarction is proportional to the size of the ligated branch. In many cases ligation of a coronary gives rise to fibrillary contractions and sudden death (Porter, Magrath and Kennedy, Kronecker); in others death may follow within a few minutes (Cohnheim and v. Schulthess-Rechberg) within an hour (Panum), or the animal may live several weeks or more (Baumgarten) if the operation is done aseptically. Death even then often occurs suddenly.

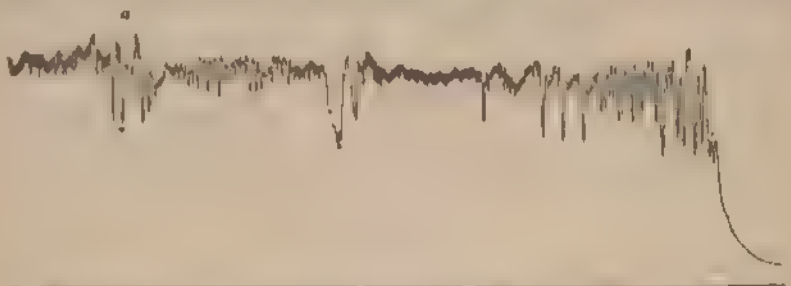

FIG. 165.—Effect of ligation of a large coronary artery upon the blood pressure. (After Cohnheim and v. Schulthess-Rechberg.) Coronary artery ligated at a.

Distribution of the Coronary Arteries.— Walter Baumgarten in Porter's laboratory was able to ligate the various coronary branches of cats and dogs under aseptic precautions and produced infarcts in the corresponding areas of the myocardium. He found the following effects by ligating the various branches:

Ramus descendens: Anterior wall of left ventricle, anterior papillary muscle, left half of the thickness of the interventricular septum.

Ramus circumflexus: Posterior wall of left ventricle apex, posterior papillary muscle, a certain extent of the right ventricle, posterior wall of left atrium, posterior third of the septum.

Ramus septi: This is given off in the dog near the origin of the ramus descendens or independently of it. Ligature produces a triangular infarction with the apex of the triangle towards the ligature.

Right coronary: Greater part of right ventricle posterior portion of the appendix atrii. (The smaller branches of the atria are not caught in the ligature.)

Baumgarten also excised the anæmic area and perfused it with defibrinated blood, and found this region was able to resume contractions when the circulation was renewed within six to eleven hours after the artery had been ligated. The region of the centre of the infarct lost its contractility before that near the periphery, indicating that a certain degree of collateral circulation, perhaps through the vessels of Thebesius, had taken place. This work explains why it is that a certain time elapsed between the obstruction of the artery and the sudden cessation of beat in the experi-

ments of Panum and of Cohnheim and v. Schulthess-Rechberg. The greater number of Baumgarten's dogs and cats survived the operation well and showed no change in heart action beyond a transitory arrhythmia lasting twenty-four to thirty-six hours. The sounds were normal in every respect and possessed no undue valvular quality. Only two animals showed signs of sudden cardiac failure one dying in the midst of violent exertion and the other soon after it, exactly as occurs in man (see page 284).

Hirsch and Spaltehols found that, though infarcts were produced by ligation of the coronary artery, the infarcts were smaller than the area supplied anatomically by the artery, and there was a not inconsiderable amount of anastomosis, especially between the branches near the surface of the heart. In man Chiari has found complete occlusion of the right coronary artery without infarction, and Pagenstecher has ligated that artery in an operation without evil result. These are the main facts regarding the coronary circulation which throw light upon the clinical conditions observed.

Pratt has shown that the excised mammalian heart can be nourished through the veins of Thebesius sufficiently to carry out forcible contractions for a considerable time, though this probably is not the case in the living animal.

It has long been a matter of debate whether the heart muscle was nourished with blood during the systolic or during the diastolic period: the earliest contention being that of Scaramucci (1689) that the coronary vessels are squeezed empty by the contraction of the heart muscle-fibres during systole and fill from the larger and more superficial coronary vessels during diastole. After a long controversy, during which Rubatel showed that the curve of coronary blood-pressure and apparently also the curve of blood velocity were exactly similar to the curve in the aorta, the question was definitely settled upon the excised heart by Porter and his pupils in favor of the old view of Scaramucci. They also found that the blood in the coronary veins is squeezed out in diastole.

The existence of vasomotor nerves for the coronaries has been proved by Manss, who found that the vagus exerted a vasoconstrictor, the accelerator a vasodilator action upon these vessels.

This was confirmed by O. Langendorff and Wiggers, who found also that adrenalin exerted a vasodilator action upon the coronary arteries of the excised heart instead of its usual vasoconstrictor action. Both Wiggers and G. S. Bond have found that the outflow through the coronary veins of the dog's heart in situ is increased by the administration of adrenalin. Bond investigated the effects of a large number of other drugs as well, and found that the coronary outflow always followed the curve of general blood-pressure, so that, under the experimental conditions, he was unable to demonstrate any specific action upon the coronary vessels, even from doses far larger than would be administered in therapeutics. However, the operation is so severe that the animals are always in profound shock.

Ida Hyde in Porter's laboratory found that the coronary blood flow was diminished by distention of the heart, a fact which may account for the weaker contraction of overdilated hearts.

SCLEROSIS OF THE CORONARY ARTERIES

PATHOLOGICAL ANATOMY.

While the sclerosis of the coronary arteries does not differ in its pathology from the sclerosis of arteries elsewhere, nevertheless the action upon the heart gives rise to clinical and to secondary pathological conditions which are quite different from those of general arteriosclerosis, and which therefore deserve special consideration.

Another important condition which is very common is arteriosclerotic or atheromatous change arising in the aorta with or without associated involvement of the coronaries themselves, but spreading so as to involve the mouths of the coronaries as they arise from the aorta, and strangulating these vessels as they pass through the aortic wall (see Fig. 166). This has the same effect as a metal band constricting an artery would have; namely, of diminishing the blood-pressure and the velocity of flow in the artery beyond it, of allowing the walls of the artery to contract down and hence of producing a further permanent secondary narrowing of the lumen, with progressive diminution in the blood supply to the part (Halsted). The course of the artery may show patches of hardening with indentations and widenings, collar-like constrictions, or uniform widenings, or, on the other hand, the arteries may be converted into uniform tubes whose walls may give the sensation of rubber tubes on the one hand (uniform fibrous sclerosis), or of absolute pipe-stems (complete calcification) on the other. This condition is, of course, particularly common in arteriosclerosis affecting the base of the aorta and in the arterio-

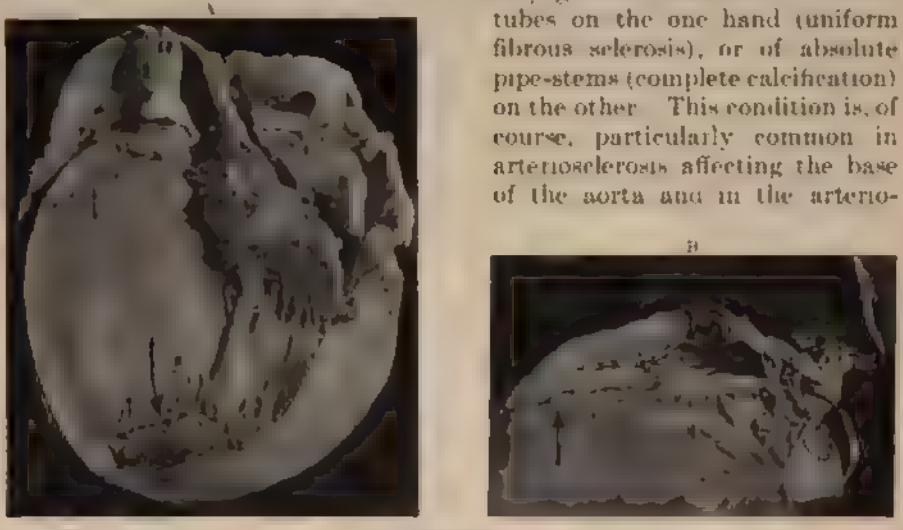

FIG. 166.—Sclerosis of a coronary artery, producing an area of infarction near the apex. A, Showing the entire specimen. B, The sclerotic coronary artery, camera brought closer, a wire has been passed through the mouth of the coronary artery.

sclerotic form of aortic insufficiency, and may account for many of the symptoms to be discussed later (see page 284).

Since the heart muscle requires much more blood when it is beating forcibly and rapidly than when it is beating slowly and quietly, it is easily seen that this collateral circulation may be sometimes adequate and sometimes not. Also, since in different individuals of the same species there are variations both in the structure and disposition of the minute arteries and in the needs of the muscle-fibres for nourishment, it is but natural that the results of coronary disease should vary greatly.

CLINICAL MANIFESTATIONS.

The clinical pictures associated with coronary sclerosis are characterized by some or all of the following features: *pain* over the precordium or down the arms, feelings of *suffocation* or of *impending death*,

paroxysms of most intense dyspnœa with palpitation, enlargement and pulsation of the liver, general weakness, sudden death.

A considerable grade of arteriosclerosis may be present in both young and old individuals without giving any symptoms whatever, as shown in the case of J. L. (page 467). Another example of this was a colored boy under the writer's care who after very vigorous life died at the age of nineteen in the fifth week of typhoid fever. Neither before nor during the fever had he had any cardiac symptoms. However, his coronary arteries were found to be very sclerotic.

Sudden Death.—Sudden death is frequently the first manifestation of the condition, and examples are almost daily in the newspapers of persons, usually men past middle life, who drop dead without warning and with no previous illness, due to sudden thrombosis of the sclerotic coronary arteries, or perhaps merely to the fact that, though the sclerotic process has been going on gradually, the instant has passed at which the cardiac nutrition becomes insufficient and ischæmia sets in with sudden functional insufficiency, just as occurs in the leg in intermittent claudication. This must be the case in many hearts in which no actual thrombosis or embolism can be found post mortem.

Paroxysms of dyspnœa such as those described on page 148, the so-called cardiac asthma, are also extremely common in coronary sclerosis, especially when combined with aortic insufficiency (vide page 366), in which case they are no doubt due to the dilatation and weakening of the left heart and the consequent accumulation of CO_2 in the blood. It has been suggested by Drs. C. M. Cooper and E. O. Jellinek of San Francisco that this was always an accompaniment of sclerosis of the right coronary artery and dilatation of the right heart, but in autopsies of two cases under the writer's care who had suffered from such attacks the right coronary was absolutely free from sclerosis.

Sensations of pain in the precordium, and especially behind the sternum, as well as pains and tenderness over various interspaces and radiating down the arms, are especially common in coronary sclerosis.

Paroxysmal Tachycardia.—Attacks of tachycardia beginning with sudden doubling of the pulse-rate and ending in sudden halving of the latter, just as is present in essential paroxysmal tachycardia, have been described by Romberg as manifestations of coronary sclerosis, and Krehl also cites similar findings.

In Romberg's case the pulse-rate rose suddenly from 100 to 200 while the respiration remained at 20. The attack lasted two days and then the pulse-rate dropped suddenly to 100. Later an aortic stenosis developed gradually and the patient died of heart failure, the autopsy showing aortic stenosis and sclerosis and marked coronary sclerosis. Dr. Barker informs the writer that he also has seen a couple of cases in which such attacks were associated with coronary sclerosis.

Quite recently similar attacks have been produced by T. Lewis upon ligating the coronary arteries in cats even after the cardiac nerves had been sectioned.

Painful sensations about the heart are particularly common in association with coronary sclerosis, but on the one hand they are by no means confined to this condition and on the other hand most extensive coronary sclerosis may be present without the occurrence of cardiac pain. The most marked form of cardiac pain, the so-called "angina pectoris" (pronounced

an'gina, not angi'na) to be discussed below, is in its most typical form usually associated with a certain degree of coronary sclerosis.

ANGINA PECTORIS

In 1768 both Heberden and Rougnon described attacks of pain in the chest. The former recognized the condition the more clearly and described it in the following words.

"But there is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned here at length. The seat of it, and sense of strangling and anxiety with which it is attended, may make it not improperly be called *angina pectoris*.

"They who are afflicted with it are seized while they are *walking* (more especially if it be *up-hill* and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life if it were to increase or to continue, but the moment they stand still all this uneasiness vanishes.

"In all other respects the patients are, at the beginning of the disorder, perfectly well, and in particular have *no shortness of breath*, from which it is totally different. The *pain is sometimes situated in the upper part, sometimes in the middle, sometime's at the bottom of the os sterni*, and *often more inclined to the left than to the right side*. It likewise very frequently extends from the breast to the middle of the arm. The *pulse* is, at least sometimes, *not disturbed* by this pain, as I have had opportunities of observing by feeling the pulse during the paroxysm. *Males* are most liable to this disease, especially such as have *passed their fiftieth* year. After it has continued a year or more, it will not cease as instantaneously upon standing still, and it will come on not only when the persons are walking but *when they are lying down*, especially if they lie on the left side, and oblige them to rise out of their beds. In some inveterate cases it has been brought on by the motion of a horse or a carriage and even by *swallowing, coughing, going to stool, speaking*, or *any disturbance of mind*.

"Such is the usual appearance of this disease, but some varieties may be met with. Some have been seized while they were standing still or sitting, also upon first waking out of sleep, and the *pain sometimes reaches down the right arm as well as the left and even down to the hands*, but this is uncommon; in a very few persons the arm has at the same time been numbed and swelled. In one or two persons the pain has lasted some hours or even days but this has happened when the complaint has been of long standing and thoroughly rooted in the constitution; once only the very first attack continued the whole night.

"I have seen nearly *a hundred people* under this disorder of which number there have been *three women* and *one boy* two years old. All the rest were men near or past the fiftieth year of their age.

"Persons who have persevered in walking till the pain has returned four or five times have then sometimes vomited . . . . The termination of angina pectoris is remarkable. For if no accident intervene but the disease go on to its height *the patients all suddenly fall down and perish almost immediately*. The angina pectoris, as far as I have been able to investigate, belongs to the class of spasmodics, not of inflammatory complaints. For

"In the first place the access and the recess of the fit is sudden.

"Secondly, there are long intervals of perfect health.

"Thirdly, wine and spirituous liquors and opium afford considerable relief.

"Fourthly, it is increased by disturbance of mind.

"Fifthly, it continues many years without any other injury to the health.

"Sixthly, in the beginning it is not brought on by riding on horseback or in a carriage, as is usual in diseases arising from scirrhus or inflammation.

"Seventhly, during the fit the pulse is not quickened.

"Lastly, its attacks are often after the first sleep, which is a circumstance common to many spasmodic disorders.

"With respect to the treatment of this complaint, I have little or nothing to advance Quiet, warmth, and spirituous liquors help to restore patients who are nearly exhausted and to dispel the effects of a fit which does not soon go off Opium taken at bedtime will prevent the attacks at night"

Heberden's contemporary, the great John Hunter, suffered from this disease, and described his attacks most vividly.

The modern aspects of the whole subject have been discussed in a masterly way by Sir W. Gairdner as well as in the more recent monographs of W. Osler and G. A. Gibson.

CHARACTER OF THE PRECORDIAL PAIN AND CLINICAL SUMMARY.

In Heberden's description we have epitomized almost all the clinical features. (1) The sudden attacks of oppression in the chest, with a feeling of strangling, and, as Hunter puts it, "as though the sternum was being drawn back to the spine," or, in the words of Matthew Arnold, as "though there were a mountain upon my chest." (2) The mental anguish (termed by Gairdner angor animi), with the fear of impending death, especially pronounced in John Hunter. (3) The intense pain, situated sometimes in the lower sometimes in upper part of the sternum, more frequently to the left than to the right (although occasionally to the latter), and very often radiating to the arm, especially the left. (4) Some of the disturbances of sensation; even Heberden speaks of numbness of the arm. (5) Changes in the pulse in some cases: intermissions, extrasystoles in some cases (Hunter); alternating pulse in others (Mackenzie). (6) The extreme pallor and constriction of peripheral arteries during the attack. (7) The sudden death. (8) The main factors in bringing on attacks, walking up-hill, flatulence and digestive disorders, bending down in undressing, mental excitement or anxiety, and especially anger, but none of the more gentle emotions, such as pity, sorrow, etc., even when felt intensely. (The effect of exposure to cold does not seem to be mentioned by these writers.) (9) The association of the condition with sclerosis of the coronary arteries. (10) Its frequent association with abnormal fatty deposits about the heart (cf. Jenner and also page 214). (11) The relief of symptoms by means of opium, warm applications, hot drinks (vasodilator mechanisms) and counter-irritation (Heberden). (12) Its incurability, owing to the seat of the trouble.

To these points clinical observations since Jenner have added (1) The existence of anginoid attacks with several conditions other than those of coronary sclerosis, particularly with over-indulgence in tobacco, with hysteria, with hyperthyroidism, and with other purely vasomotor phenomena, as well as with practically all the valvular diseases of the heart. (2) The frequent association of angina pectoris with certain definite areas of tenderness which represent spinal segments corresponding to the referred pain. (3) The occurrence of rise in blood-pressure with each attack. (4) The relief of the attacks by inhalations of amyl nitrite and other vasodilator drugs.

Sir William Gairdner has called attention to the occurrence of certain cases resembling Heberden's angina pectoris in every way except in the absence of pain as a symptom (angina sine dolore).

Paths Traversed by the Pain Sensations.—The afferent impulses from the heart have been traced by Ludwig and Cyon through the depressor fibres of the vagus. It has been shown by Eyster and Hooker that the afferent impulses from the aorta and coronary arteries do not take this same path but pass upward in the main bundle of the vagus. There is no evidence from animal experiment that afferent impulses pass in any other way, but Henry Head, as a result of his most extensive studies upon pain in visceral disease, states that this "produces impulses which pass into the spinal cord by the white rami. The segment on which they infringe is excited and pain is produced. At the same time all potentially painful influences passing into this segment from the afferent nerves are exaggerated, and ultimately the body wall may become tender."

These sensations of referred pain follow the same path as has been described by Bayliss for the vasodilator fibres with which protopathic sensation seems to be closely associated, as shown in herpes zoster etc. Protopathic sensations are referred back to the distribution of the corresponding nerve segments without close reference to the points at which they arise.

Insensibility of the Heart to Touch.—The heart itself seems to be devoid of tactile sensation for Harvey gives the following description of the condition in the nineteen-year-old son of Viscount Montgomery, who had a fistulous opening in the chest wall over the heart following fracture of the rib in early childhood. "I found a large open space in the chest into which I could introduce three of my fingers and my thumb . . . I saw that I was handling the apex of the heart covered over with a layer of fungous flesh by way of external defence, as commonly happens in old foul ulcers. . . . The youth never knew when we touched his heart except by the sight or the sensation he had through the external integument."

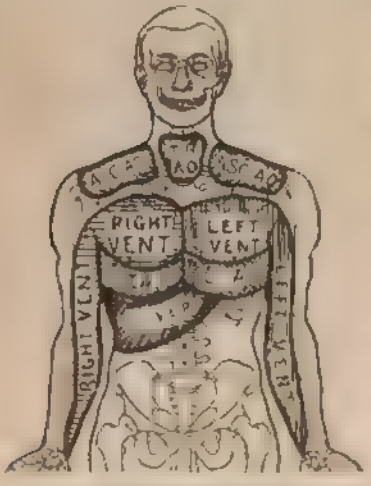

Fig. 167.—Distribution of pain in attacks of angina pectoris. So [illegible] after Head [illegible] Mackenzie. [illegible] area corresponding to the ascending aorta. TR. AO. area corresponding to the transverse aorta. L. A. R. A. area corresponding to left and right auricles.

Palpitation and Anginal Sensations Compared.—The sensations which may be felt from the heart itself may be either rhythmic and felt as a distinct sensation **accompanying each systole of the heart,** such as the feeling of palpitation, or the pain felt at each beat in some cases of pericarditis, especially those associated with pneumonia. The sensation in the latter condition may, however, arise in the parietal pericardium, and may have nothing to do with the heart itself.

Sensations of palpitation may be very distressing, partly on account of the feelings of suffocation which accompany **them, partly on account of the mechanical** shock of the heart beating forcibly against the chest wall like a bird in a cage. But, however intense and distressing, the sensation of palpitation is always a pressure sensation and never one of pain. On the other hand, the real cardiac pain is never intermittent, never felt as a distinct sensation with each beat of the heart, but, whether dull and aching or sharp and stabbing, it has no throbbing quality about it. It is, therefore,

not at all homologous with the sensation of palpitation and must belong to a quite different category. Hirschfelder has added some evidence for this view by observing that in some cases of palpitation the sensation was referred definitely to the root of the aorta, and was exactly similar in character to other sensations of throbbing in the radial artery alone, which were sharply localized along its course and not spreading like a protopathic sensation.

Referred Pains in Angina Pectoris.—James Mackenzie and Henry Head have called attention to the commonness of referred pain and tenderness in angina pectoris. Mackenzie showed that there is often tenderness in the areas supplied by the second and third cervical segments, whose fibres along with some from the spinal accessory run down to the heart through the vagus. This would account for the occipital headaches and tenderness of the sternocleidomastoid and trapezius muscles which are frequently present. The muscular tenderness is elicited by squeezing gently between the thumb and forefinger.

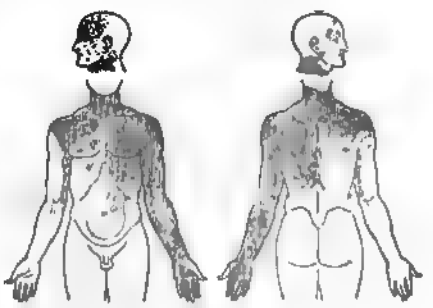

FIG. 168.—Distribution of attacks of pain and sensory disturbances in a case of angina pectoris. (After Head, with permission of the publishers of Brain.)

The distribution of the pain and hyperæsthesia, according to Head, bears a close relation to the chamber most affected, and particularly to the somatic segment of the embryo to which it corresponds.

	Correspond embryologically to	Nerve supply	Associated phenomena and pain referred to
Auricles	5, 6, 7, 8 thoracic	5, 6, 7, 8 segments	Lower axilla and shoulder-blades.
Ventricles	2, 3, 4, 5, 6 thoracic	2–6 thoracic segments	Chest wall from 2d-7th rib, ulnar surface of forearm to wrist, and inner aspect of upper arm.
Ascending aorta . .	3 and 4 cervical	3 and 4 cervical segments	These segments also to 3 and 4 c. and 1 thor. Tenderness in neck of sternomastoid and trapezius muscles. Tenderness and pain at back of neck. (Dilatation of pupil?)
Transverse arch . . .	C. IV.	C. IV.	Laryngeal areas of neck (4th branchial bar).
Descending aorta	Thoracic segments corresponding 2–12	2–12, esp. 4 12	Back or front of chest, especially below nipple; abdomen.
Pulmonary artery	C V–VIII. . .	C. V. . .	Outer two-thirds of arm and hand; arm muscles.

Thus, the auricles (atria), which are the hindmost in the development of the cardiac tube, receive their innervation from and refer their pain to the fifth, sixth, seventh, and eighth thoracic segments. The ventricles, the next chambers headward, correspond to

the second to the sixth thoracic; the ascending aorta from the semilunar valves to the origin of the ductus arteriosus corresponds to the primitive aorta with the third and fourth branchial artery, and the pain is referred to these segments (but an aneurism, etc., involving this in adult life will also involve the neighboring nerves and the pain will be referred to the first, second, and third thoracic segments as well). The fifth to the eighth cervical segments, corresponding to the pulmonary artery, will not be involved, and pain may not be referred over these areas.' (There are many notable exceptions to this rule even among Head's cases; but there is usually overlapping of these areas.)

Sudden Death and Motor Disturbances. — The phenomena thus far considered are purely sensory; and the question arises, what are, if any, the motor disturbances connected with angina pectoris? It is evident that the cessation of the heart-beat in sudden death that occasionally occurs may be due either to the occlusion of the artery or to a sudden onset of complete heart-block as in the Adams-Stokes syndrome. The latter condition is sometimes associated with angina pectoris and very frequently with coronary sclerosis (see page 472), though this is rare, and more frequently the pulse becomes regular after a short time, or sudden death from heart-failure sets in just as in the experiments of Cohnheim and v. Schulthess-Rechberg.

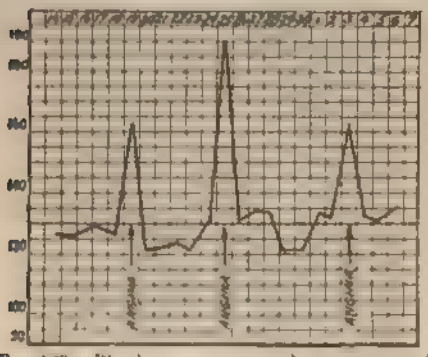

FIG. 169.—Blood-pressure curve showing crises of hypertension during attacks of angina pectoris.

During the attacks of angina pectoris the blood-pressure is often high, though Mackenzie states that in many cases there is no change whatever. This seems to be due to a true pectoral vasomotor crisis in the sense of Pal, rising sharply with and falling sharply after the attack, as shown in Fig. 169

ETIOLOGY AND VARIETIES OF ANGINA PECTORIS.

The idea that sclerosis of the coronary arteries was the lesion which caused angina pectoris seems to have originated not with Heberden but with Edward Jenner, the discoverer of vaccination, who was so certain of its pathology that before doing an autopsy upon a case he made a bet with a friend that he would find thickening of the coronary arteries. He won the bet.

This indeed seems to be correct for almost all cases of fatal angina, since Huchard found coronary sclerosis present in 128 out of 145 autopsies recorded in the literature, and most of the others were in cases of adherent pericardium or valvular disease. A few cases of death have occurred in attacks of angina due to tobacco or in post-febrile conditions where the coronary arteries were clear, but in these the possibility of obscure myocardial change must be borne in mind (Osler).

[1] His own cases, no. 37 and 38, as well as cases of many other writers, show tenderness during and after attacks due to aortitis, involving the ascending aorta.

Angina Pectoris without Coronary Sclerosis.—However, in 1812, J. Latham reported a number of cases which, in spite of the occurrence of intense anginal symptoms, did not run the usual course ending in sudden death, and to these he gave the name of "pseudo-angina" (angina notha).

Bean, Stokes, and Graves also described reflex and toxic forms of angina, but a much clearer light was thrown upon the subject by Nothnagel's article entitled "Angina pectoris vasomotoria." He says, "We must interpret this symptom-complex to indicate that we are not dealing with a disease which arises primarily in the heart, but that the symptoms of stenocardia are of secondary origin and are brought on by a very general spasm of the arteries."

The term "pseudo-angina" has been severely criticised by Balfour and Gibson, since "angina" is a symptom, not a disease, and in all cases it is a very real one. Nothnagel's term, "vasomotor angina," or Huchard's "reflex angina," seems to the writer to be preferable.

Theories as to Causation of Anginal Pain.—Many theories have been advanced to explain the causation of pain in anginal attacks. These may be classified as follows:

(1) **Ischæmia from Coronary Stenosis.**—The original view of Jenner was later supplemented by Allan Burns, that the attack may be brought on by asphyxia of the heart muscle when there was a disproportion between the amount of blood flowing to it and the amount of blood which it needed. Potain, in 1870, was the first to introduce the theory that angina pectoris is due to "the intermittent claudication of the heart", but Allan Burns had already completely demonstrated this causal factor in 1809 and had described his observations in the following words: "If we call into vigorous action a limb round which we have with a moderate degree of tightness applied a ligature, we find that the member can only support its action for a very short time, for now its supply of energy and its expenditure do not balance each other . . . we witness an indication of an extreme degree of debility and we have the patient complaining of an unusual painful feeling in the limb, but still all its muscles are in a state of inactivity. . . . If a person with the arteries of the heart diseased in such a way as to impede the progress of the blood along them attempt to do the same (ascend a steep or mount a pair of stairs), he finds that the heart is sooner fatigued than the other parts are," and the same pain results.

(2) **Ischæmia** from vasoconstrictor spasm of the coronary arteries, which reduces the functional condition to the same state as described by Allan Burns for the coronary sclerosis. This seems to apply to the vasomotor and toxic anginas and often constitutes a factor superimposed upon the coronary sclerosis in the angina vera. Such an action of drugs upon the coronary vessels has been demonstrated on the excised heart by O. Loeb, Langendorff, and Wiggers (see page 281).

(3) **Acute dilatation** of the heart, producing a pain similar to that of intestinal colic. This theory particularly has been adhered to by many writers. The similarity between the anginal pain and that of renal, biliary, pancreatic, and intestinal colic suggests that it belongs to the common form by which the visceral nerves give expression to overdistention. Some dilatation usually accompanies the attack, and seems to be a primary cause of the pain in cardiac overstrain and in many cases of valvular lesion.

(4) **Neuritis.**—It may at times be due to neuritis of the cardiac nerves, or, on the other hand, to a neuritis primary in the brachial nerves and referred to the heart. Lesions of the cardiac plexus have been described by Lancereaux, Grocco, and Benenati, but Henrard and others have failed to find them. Nevertheless it is quite possible that substances like tobacco (nicotine), which stimulate sensory nerves in the heart and which have a specially toxic action upon the ganglion cells, may produce toxic neuritis of these nerves.

(5) **Neuralgia** of the cardiac nerves.

(6) Action of other constitutional diseases like gout, diabetes, and chronic nephritis.

But it is most probable that the effects are due to the other above-mentioned factors which accompany these diseases—arteriosclerosis and the presence of vasoconstrictor substances either as retention products or internal secretions.

ASSOCIATED CLINICAL CONDITIONS.

The various conditions with which angina is associated most commonly might be classified as follows:

I. Organic Lesions.
- A. Sclerosis of coronary arteries.
- B. Aneurism, especially of first part of ascending aorta.
- C. Valvular lesions, especially aortic insufficiency. (This constitutes a very common group.)
- D. Aortic aneurism, especially of the sinuses of Valsalva and the ascending arch.
- E. Adherent pericardium. (The most frequent form which is seen in children.)

II. Vasomotor anginas.
- A. Hysterical type, most common in women, associated with other vasomotor disturbances and stigmata of hysteria.
- B. Toxic due to the action of various poisons, especially (*a*) tobacco, (*b*) caffeine, taken both as tea and as coffee.
- C. Associated with hyperthyroidism and exophthalmic goitre.

III. Attacks of more or less anginoid pain occur in the cases of acute dilatation of healthy hearts, due to primary cardiac overstrain.

Angina Pectoris in Valvular Diseases.—The attacks of angina pectoris associated with coronary sclerosis, which represent the original form described by Heberden, are usually designated as angina vera. These are very often associated with valvular lesions, especially with aortic insufficiency in which the coronary lesions are usually continuous with those of the aorta, but they are also common in association with other valvular lesions, since it is rare to find a case of chronic valvular disease without some disease of the coronary arteries. The presence of valvular disease, therefore, rather favors than excludes the diagnosis of coronary sclerosis.

In spite of the frequency with which these two conditions are associated, occasionally one encounters cases of angina with valvular disease, especially aortic insufficiency, without any disease of the coronary vessels whatever, as was well exemplified by a patient with a ruptured aortic valve who was for five years under observation at the Johns Hopkins Hospital. During this time he suffered from very frequent attacks of typical angina pectoris. He died suddenly while at stool. Autopsy showed rupture of aortic leaflet. The coronary arteries were soft and the walls were not thickened anywhere.

Angina Pectoris in Acute Dilatation.—It is possible: (1) that under these conditions acute dilatations of the heart, due to momentary diminution in tone of the heart muscle, might be the immediate cause of the pain, which would thus be of primary cardiac rather than vascular origin. (2) That in such dilatation, etc., centripetal stimuli may arise in the heart which may cause a general vasoconstriction. (This is contrary to the usual depressor effect of stimuli arising in the heart, but it is not at all certain that in the presence of such a pathological condition as angina pectoris the paths of least resistance in the central nervous system may not be quite

different from what they are in the normal individual.) (3) Miss Hyde in Porter's laboratory has shown that dilatation of the heart in itself caused diminution in the flow through the coronary arteries, and it is possible that the circulation may thus be diminished to a point at which relative ischæmia of the heart may set in and cardiac pains result.

Angina Pectoris in Aneurism.—Attacks of angina pectoris are very common in cases of aneurism involving the ascending arch, and especially in early small aneurisms near the sinuses of Valsalva. This has long been known, but is the subject of an especially interesting article by Dr. Osler upon "Angina pectoris as an early symptom of aneurism."

The anginoid pains in this condition are probably simply reflex, not the result of primary peripheral vasoconstriction, cardiac ischæmia, etc., but simply the occurrence of pain sensation arising in the aortic walls from overstretching of the aorta under pressure heightened from any cause whatever, or from increased excursion of the aortic wall as a result of increased systolic output, etc., as is so frequently seen in the abdomen in nervous women with epigastric pain due to a throbbing of the abdominal aorta. In the later stages of the aneurism, the symptoms may be less intense, due perhaps to the fact that by erosion, etc., pressure upon the aneurism has diminished, perhaps to the fact that after a time endings of the sensory nerves have been permanently injured or rendered less sensitive by the progressive change in the aortic wall.

Anginal Attacks in Children.—Angina pectoris also occurs in children, especially in association with mitral stenosis as illustrated by the following case. The patient was a boy aged 8 who had had rheumatism in the right hip two years previously, and since then "had attacks of pain over the heart, especially after exercise. The pain was so severe that it compelled him to stand perfectly still until it passed off, his cheeks became blue and pale. He sometimes felt as though held in a vise, but never had any feeling of fear. He also had at times pain on the r i g h t side over about the sixth rib, which was sometimes present with that on the left side, but often present without it. Exercise seemed to bring on both. Examination showed a very slightly enlarged heart with systolic retraction over the fourth left interspace, none about origin of diaphragm (Broadbent's sign absent). Area of cardiac flatness changes with respiration. The first sound at the apex was snapping in character and was preceded by a well-defined rumble. Second sound was clear, accentuated over the pulmonic area. Pulse 92 per minute, of good volume, regular in force and rhythm."

Such attacks are quite definite angina vera in the sense of Heberden, and indeed the latter includes a similar case in his list. In children the association is, however, much more commonly with valvular lesions than with coronary sclerosis, and perhaps most frequently of all with

Adherent Pericardium.—This is an extremely common concomitant and cause of anginal attacks, especially in children and adolescents. The pains are, perhaps, simply reflex aches from the ordinary tugs upon the pericardium, perhaps brought about by the stretching of the pericardial fibres which occurs when the heart becomes dilated.

VASOMOTOR ANGINA.

The second great group of cases with anginal symptoms are those in which the anginal symptoms are of purely vasomotor origin (Raynaud's disease of the heart) and are not associated with organic lesions,—the angina pectoris vasomotoria of Nothnagel (angina pectoris spuria of Latham, angines de poitrine reflexes of Huchard). The characteristic phenomenon in this group is the occurrence of g e n e r a l o r l o c a l v a s o c o n s t r i c t i o n u s h e r i n g i n t h e a t t a c k; that is, there are usually coldness, numbness, often tingling, weakness, and heaviness in the left arm, pallor of the latter, with marked diminution in size and caliber of the left radial, often also of the right radial artery, sometimes of the vessels

of the leg, trunk, and head. The patient may become pale and blue or the lips ashen, and the course of the attacks may exactly simulate those of coronary sclerosis. Death in such attacks is, however, extremely rare. It has occurred in several cases in which no coronary sclerosis nor other lesion was present to account for the death. However, Dr. Osler suggests that in these cases there may have been myocardial changes demonstrable only by the method of Krehl.

Hysterical Angina.—The most common form of vasomotor angina is the neurotic or hysterical type, which is most common in young women and is associated with the other stigmata of hysteria,—exaggerated emotional response with marked histrionic tendencies, transitory vasomotor disturbances, shifting areas of anæsthesia and hyperæsthesia, characteristic epileptiform seizures, and the existence of hysteriogenic zones where pressure calls forth the above-mentioned symptoms.

Clinical Groups with Anginal Symptoms and their Characteristic Features (modified from Huchard).

Coronary Angina.—*Site of disturbance.*—Stenosis or obliteration of the coronary arteries. (In some cases valvular lesion or aneurism only). *Age.*—Age of arteriosclerosis after 40. *Factors bringing on attack.*—Effort of some sort, mental or physical. Rarely spontaneous, sometimes nocturnal. Not associated with any other form of neurosis. *Nature of pain.*—Agonizing sensation of pressure. Usually felt most acutely behind sternum. Referred pain down arm, especially left arm, and over chest, neck, etc. *Duration.*—2 to 15 minutes, stopping soon after standing still. *Attitude.*—Silent, immobile. *Prognosis.*—Grave; almost always fatal. *Treatment.*—Vasodilators.

Hysterical Angina.—*Site of disturbance.*—Central nervous system acting through the vasomotor nerve and cardiac plexus. *Age.*—At all ages, even childhood; sometimes at menopause. Most frequent in women. *Factors bringing on attack.*—Usually spontaneous onset without effort, often recurring at fixed hours and associated with other neurotic symptoms. *Nature of pain.*—Pain less agonizing, with feeling that the heart is distended—felt most intensely at the apex. *Duration.*—1 to 2 hours, not diminished by standing still, not increased by walking. *Attitude.*—Agitated; walking about. *Prognosis.*—Mild; never fatal. *Treatment.*—Antineurotics and antineuralgics.

Gastro-intestinal.—*Site of disturbance.*—Distention or neuralgia due to gastric troubles. *Age.*—At all ages, especially among women. *Factors bringing on attack.*—Not brought on by effort. *Nature of pain.*—Precordial, not substernal pain; with fulness of chest and distention of heart but less radiation. Signs of dilatation of right heart; increase of inverse diameter to right. *Duration.*—1 to 2 hours. *Prognosis.*—Death rare. *Treatment.*—Antidyspeptic remedies.

Tobacco.—*Site of disturbance.*—Spasm of coronary arteries. *Factors bringing on attack.*—Angina associated with toxic disturbances, vertigo, gastric and respiratory troubles. Onset spontaneous. *Nature of pain.*—Attacks associated with bradycardia, intermittent pulse, arrhythmia, palpitations. Attacks longer than those of angina vera. *Prognosis.*—Death

rare. Attacks often disappear rapidly on giving up tobacco. *Treatment.* Stopping tobacco, tea, and coffee. Rest and mental quiet. Light diet. (Anginas due to tea, coffee, etc., brought about by the same cause.)

Acute Cardiac Overstrain (with or without Valvular).—*Site of disturbance.* Sudden dilatation of the heart. *Age.*—At any age, but most common in young athletes, soldiers, anaemic girls. *Factors bringing on attack.* Comes on in the midst of some unusual effort, such as a mountain climb, boat race, a charge, or a dance. *Nature of pain.* In the heart itself, usually retrosternal. Associated with signs of dilatation to right and left, extreme dyspnœa, often systolic murmur and arrhythmia. *Duration.* In maximum intensity a few minutes, after cessation of attack, the pains often continuing or recurring as less intense pain, tachycardia or arrhythmia usually persisting some time after attack. *Attitude.*—Immobile. May throw himself to the ground in the midst of the effort. *Prognosis.* Death rare. Permanent weakening of the heart if the over-exertion is soon and frequently repeated. *Treatment.* Prolonged rest and general cardiac therapy until cardiac dilatation has passed off; gradual resumption of active life.

Angina Pectoris in Hyperthyroidism.—Very closely resembling the neurotic group are the cases of angina associated with exophthalmic goitre, in which the attacks are sometimes more like those of neurotic, sometimes more like those of the coronary type. The crucial point in the diagnosis is the detection of hyperthyroidism by the application of the numerous tests for Graves's disease, etc.

A case which has been for the past year and a half and still is under the writer's care will serve as type of this condition (see page 586).

Treatment is the same as for the Graves's disease which is the primary condition (see Part IV, Chapter II). The attacks themselves may be treated symptomatically with amyl nitrite, etc., but the important factor is the treatment of the underlying disease.

Tobacco Angina.—Anginal attacks due to tobacco are not uncommon, both in young persons beginning their first excesses in tobacco and in older persons whose over-indulgence is adding itself to a beginning or advancing coronary sclerosis. In both the symptoms disappear soon after the tobacco is absolutely given up, persistence of the attacks more than a few days after this being evidence that some damage to the coronaries has occurred. The attacks themselves may very closely resemble those of true angina, but very frequently precordial pains not of an anginal character may be felt by smokers between or for some time before such attacks.

The main factor in the effect of tobacco smoke, as shown by Ratner and Lee, is the nicotine, although small amounts of HCN, CO, and pyridine bases are present in the smoke. Moreover, it is probable that the action of smoked tobacco is exerted especially upon the coronary arteries, because it enters the heart directly from the pulmonary vein without preliminary dilution in the peripheral circulation.

Nicotine seems to have the effect of (1) stimulating the vagus, 2) producing vasoconstriction (3) thereby of raising the blood-pressure. In most cases this leads gradually to hypertrophy of the heart, but in some, especially weaker individuals, it tends to facilitate dilatation, thus facilitating angina. Moreover, Jackson and Matthews have recently shown for aconite, which in many ways is a similar drug, that much of its action is exerted through stimulation of the sensory endings of the depressor nerve. It is possible that nicotine angina is due in part to similar sensory stimulation.

Angina in Acute Dilatation.—The attacks of pain and precordial discomfort during acute cardiac overstrain and dilatation may reach anginoid intensity, as was noted by da Costa among the soldiers of the Civil War. He not infrequently encountered patients who had suffered so intensely in the midst of a charge that they could endure it no longer and had thrown themselves to the ground, exposed to almost certain death from the point-blank fire of the enemy, rather than continue to bear the torment within (page 124). These pains are usually retrosternal, often with numbness of the arms and tingling in the fingers, and associated with feeling of compression and with palpitation. Although they occur in the midst of extreme effort and would scarcely be confounded with angina pectoris vera, yet, since angina pectoris is a symptom and not a disease, these cases must be classed along with it.

DIAGNOSIS.

The actual differentiation of the various groups is not always easy in the individual cases, as one frequently has a coronary sclerosis with a tobacco angina superinduced upon it, a gastric etiology where there are already attacks of angina vera, etc., and since it is a safe rule never to diagnose the milder conditions until the more serious can be ruled out with reasonable probability. These cases may cause the physician anxiety, since he remains uncertain whether to expect sudden death or whether he is dealing with a comparatively mild condition.

Case Illustrating Doubtful Diagnosis.

E. W., widow, aged 65, has had since her menopause at 54 occasional attacks of precordial pain, most intense just behind the sternum and especially about the level of the third costal cartilage. She feels as though some one were boring through from sternum to spine with a sharp instrument. The pain is also felt over the left side of the chest and down the left arm, which sometimes becomes numb, cold, weak and heavy. During the attack she feels as "though the end has come." These attacks come on apparently spontaneously without definite association with either emotional disturbance, exposure to cold or muscular effort. They last an hour or two and are relieved by amyl nitrite or nitroglycerin. She feels weak for a day or so after an attack, but at other times is extremely active for her age and rarely short of breath. The patient is not at all neurotic. She has used coffee and beer in moderation all her life. It must be added that near the end of the menopause and before the first cardiac attack she had a severe spell of grippe which kept her in bed for four weeks and left her very much prostrated.

On physical examination the patient is well nourished. Slightly emphysematous, but lungs otherwise normal. Heart not enlarged, action regular in force and rhythm; sounds clear, neither second sound especially accentuated. Pulse between attacks is of good large volume and quality, apparently under normal tension, vessel wall not specially thickened. No ascites. Liver not enlarged. Feet always swollen from varicose veins, not especially so during or after attacks.

In this case the question of crucial importance is whether the angina is due to the occurrence of the menopause and is neurotic, or to the influenza which she contracted about the same time and which may have brought on a coronary sclerosis. The attacks themselves resemble angina vera, although their duration is longer than usual. The age of the patient and the history of severe influenza also are in favor of coronary sclerosis. On the other hand the fact that ordinary exertion does not seem to bring them on, but that they occur when the patient is moderately quiet, is in favor of the neurotic. It must, however, be borne in mind that the patient's statements in this regard

may be inaccurate, and, further, that in occasional cases, where the diagnosis of functional angina seemed quite well established, autopsy has shown definite coronary sclerosis. It seems impossible to establish a definite diagnosis here, and the management of the case is therefore directed toward the severer form, ordering as quiet a life as the patient will carry out (since potassium iodide is not well borne), vigorous use of amyl nitrite and nitroglycerin at the time of the attacks, and erythrol tetranitrate thereafter. A diet of small quantities of food low in purin bodies and salt is insisted on.

Since these measures have been instituted she has remained entirely free from anginal attacks for over two years, in spite of another attack of influenza. These facts are in favor of a reflex origin of the condition.

Differentiation from Abdominal Diseases.—Angina pectoris is, as a rule, easily differentiated from other diseases, though occasionally an attack of biliary, pancreatic, or left renal colic referred to the shoulders or even intestinal colic high in the epigastrium may closely simulate it. Careful physical examination and location of the areas of tenderness over the affected viscus should rule out this error.

TREATMENT.

General Therapeutic Measures.—The old treatment of Heberden mentioned above,—"quiet, warmth," and hot drinks, even if spirituous, also "opium," best in the form of morphine, 15 mg. (¼ gr.) hypodermically or by the mouth, during the attack, and repeated if necessary. As Heberden stated, it is well to bring on perspiration (and hence vasodilatation) in any way possible.[1]

Nitrites.—The most important means for the relief of the attack is, however, the inhalation of amyl nitrite.

In 1867, Lauder Brunton tried the effect of inhalations of this substance upon patients suffering from an acute attack of angina pectoris, and demonstrated that it produced very marked, almost instantaneous relief. He was led to investigate this substance by the realization that the attack was accompanied by vasoconstriction and high blood-pressure, and by the knowledge that the newly investigated amyl nitrite had been found to have a vasodilator action. Lauder Brunton's observations have been generally confirmed, and this drug has become the classical remedy for relief of the attack. Its action should be supplemented at once by hypodermic or oral administration of one or two drops of spirits of nitroglycerin (or more if the patient has been found resistant to it), and this may be followed by erythrol tetranitrate by the mouth, since this drug exerts a slower action lasting over three to six hours. Erythrol tetranitrate should be continued for some time after the attack.

Potassium Iodide.—Between attacks potassium iodide in moderate doses—0.3 Gm. (gr. v) to 4 Gm. (℥i) t.i.d.—should be given, as it seems to diminish the frequency and severity of attacks.

Dr. G S. Bond in the writer's laboratory has found that practically all the drugs which he has investigated affected the outflow from the coronary veins in the dog's heart exactly as they affected the general blood-pressure. Amyl nitrite and nitroglycerin were no exceptions to this rule. They lowered the general blood-pressure and decreased the outflow through the coronaries The effect was the same whether the heart was dilated or not, and seemed also to be independent of the strength of the heart. In view of these findings, it must be borne in mind that Hewlett has found that a rise in blood-pressure follows quite uniformly within one minute after the inhalation of amyl nitrite is begun. Whether it is the fall of blood-pressure or the rise of blood-pressure which is accompanied by increased flow through the coronary vessels after the inhalation cannot be regarded as

[1] Perhaps this may bring with it a dilatation of the coronary arteries.

certain. It must be admitted, however, that in Bond's experiments the coronary arteries were not in a state of vasomotor spasm, and therefore the analogy is not an absolute one.

It is also probable that the mere lowering of the general blood-pressure, independently of any action upon the coronaries, tends in itself to relieve the cardiac dilatation by diminishing the work of the heart.

Caffeine, Theobromine, and Theophylline.—Caffeine and especially theobromine and theophylline preparations—especially acettheobromine sodium ("agurin") and acettheophylline—have been highly recommended, from the clinical stand-point, by Askanasy, Kaufmann and Pauli, R. Breuer, Buch, Pineles, v. Leyden, and others, to relieve and to ward off the attacks of stenocardia.

Oswald Loeb has given an experimental basis to these observations by demonstrating on the excised heart that these drugs increase the blood flow through the coronary vessels as well as increase the systolic output and the force of the heart-beat.[1] Theobromine and theophylline are to be preferred to caffeine, since they do not increase peripheral resistance and have little action upon the higher nervous centres, but, on the other hand, a more marked action on the coronary arteries. On the other hand, they are not very certain in producing their effect. They may be helpful in some cases and may absolutely fail in others; and, while they are worthy of a trial in almost every case, they cannot as yet be expected to supplant the nitrites and iodides.

Diet.—Diet is all-important. It should be chiefly *lactovegetarian* in character. The *meals* should be *small* in amount, to prevent overloading and distention of the stomach and hence the pushing up of the diaphragm. *Gastric fermentation* should be prevented by removing from the diet any articles, such as soft hot breads, heavy and greasy pastry, etc., which may be found to produce flatulence, and by general treatment of the gastric condition. *Air-swallowing* should be carefully looked for and treated (see page 604). *Meat* and *soups* should be reduced to small quantities, since they contain considerable quantities of purin bodies which have a vasoconstrictor action and which also act injuriously upon the kidneys. The vegetable and cereal foods should make up the bulk of the diet. Salt should be reduced for the same reason. Liquids should be restricted to about 1500 c.c. a day.

Milk may be a staple article in the diet, unless, as in many persons, it tends to flatulence. This is sometimes obviated by adding a very little weak tea or coffee, but very often it must be dispensed with altogether.

Tobacco should be absolutely excluded in both organic and functional cases.

Tea and coffee in small amounts (one cup a day, very weak) probably have very little effect upon the average individual who has been accustomed to them, but may be quite important factors in bringing on the attacks in persons whose sensitiveness is a little above normal and in whom there is a tendency to angina. It is best for them to be given up.

Local Treatment of the Chest Wall. Vigorous counter-irritation to the chest wall, by blistering, etc., is also of value, and Hasselbach and Jacobaeus report very marked improvements, lasting a year or so, from

[1] Dr. G. S. Bond, in the writer's laboratory, has been unable to produce any appreciable change in the outflow from the coronary veins of the dog's heart in vivo, and with the amyl nitrite and nitroglycerin observed a marked decrease in the outflow, even when the animal's heart was dilated.

exposure of the precordium to the Finsen light for an hour a day until a marked cutaneous reaction or even blistering has set in.

Electrical Treatment.—J. O. Hirschfelder states that in five cases he has obtained striking relief of the symptoms by treatment with the galvanic current, applying the anode (a pad 4 cm. in diameter) to the neck over the course of the vagus, and the cathode (6–12 cm. in dianeter) to the precordium, and passing a current of 20 milliamperes for five minutes to each side of the neck. One patient remained free from attacks until his death two years after the treatment; another has remained free for several years. In the other three the relief was less permanent, but still very gratifying.

In other cases the use of electric baths, and especially with the sinusoidal current, may be of value (Rumpf), but the effect is readily overdone. The alternating current is certainly much less soothing than the sinusoidal.

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PART III.

I.

ENDOCARDITIS.

In spite of the greater frequency of arteriosclerosis and myocarditis, the clinical pictures of valvular diseases are so much more definite as to render them the most striking of all diseases of the heart. They constitute indeed a large percentage of all diseases seen by the physician, numbering 1781 (7.6 per cent.) of the 23,200 cases admitted to the medical service of the Johns Hopkins Hospital from 1889 to 1908.

HISTORICAL.

Vieussens in 1715 described lesions of the valves occurring in the form of warty or cauliflower excrescences or vegetations, which prevented the closure of the valves. Virchow called attention to the fact that as a rule these vegetations were not situated at the margins of the cusps, but at a little distance from the margin, at the line of closure where the cusps struck together, at the point where injury to the endothelial cells was most likely to occur. That this injury was usually due to the action of bacteria was shown when Winge and Heiberg and Virchow in 1869 demonstrated macroscopically the presence of minute granules within the vegetations. In 1883 Weichselbaum cultivated staphylococci and streptococci from endocardial vegetations, and his pupil Wyssokowitch, as well as Orth and Ribbert, produced them experimentally in animals by the injection of bacteria into the blood.

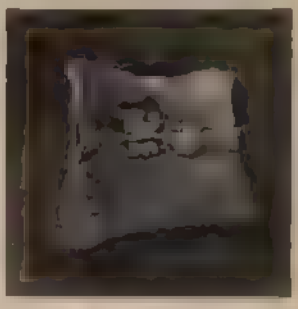

Fig. 170.—Fibrinous deposit upon an aortic cusp one hour after mechanical injury to the valve [illegible] cauliflower-shaped mass of fibrin tends to plug the hole in the valve. (Bulletin of the Johns Hopkins Hospital Bulletin.)

PATHOLOGICAL ANATOMY.

Development of the Lesions.—Mechanical or toxic injury is an important factor in bringing about these lesions upon the valves. Indeed Wyssokowitch found that his experiments succeeded only after he had punctured or injured the valves with probes; while Ribbert supplied the mechanical factor by injecting emulsions of potato cultures which contained small masses of potato that hurled themselves against the valves. Prudden, on the other hand, found that infection of the valves took place in much the same way if the valves had been injured with chemical substances. The fibrinous exudate is exudated rapidly after the injury, and is whipped into strands by the action of the current, so that within one hour after mechanical injury of the aortic valve a mass of fibrin having the cauliflower shape of a vegetation may be found filling the hole in the valve (Hirschfelder).

Ulcerative Endocarditis.—The fate of this fibrinous exudate and the type of the lesion varies with the virulence of the germ. If the virulence is high the lesion is often large and may involve the walls of the auricle or ventricle (mural endocarditis) as well as the cusps of the valve (valvulitis). The necrosis spreads into the deeper tissues of the valve or even penetrates through it, and the vegetation consists of a mass of degenerated fibrin, clumps of bacteria, and necrotic tissue (Fig. 171) rich in polymorphonuclear leucocytes. Under the influence of the ferments which these secrete, the masses become partly liquefied, so that their attachment to the cusps is loosened and they may be readily swept off as emboli by the force of the blood stream only to cause infarction and abscesses in distant tissues. Such emboli naturally vary in size from a small bit of fibrin barely capable of plugging a capillary to a mass almost the size of the valve itself. However, they rarely reach the tremendous size attained by the non-septic emboli which arise from intra-vitam thrombi in the auricles.

FIG. 171.—Mitral endocarditis showing large vegetations. *A* mitral portion of the vegetations, *B* vegetations along line of closure.

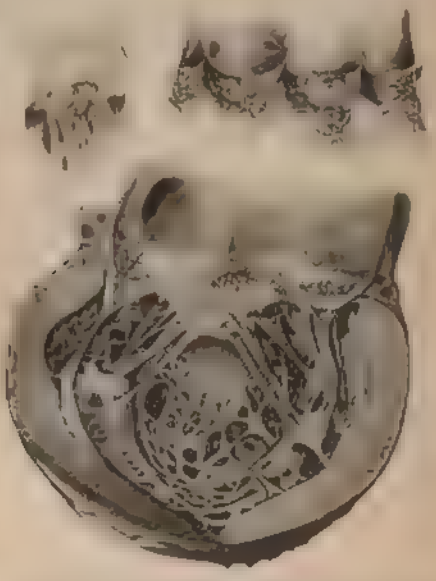

FIG. 172.—Injection of chronically inflamed valves. After v. Langer.

Chronic Endocarditis. When the bacteria upon the valves are less virulent or the immunity of the patient develops, a different process occurs. The areas of necrosis are smaller and are walled off with leucocytes. Later these give place to the fibroblasts and plasma cells of chronic inflammation, which in turn are replaced by strands of newly formed connective tissue, which push out into the exudate and finally replace it altogether, leaving a solid vegetation composed entirely of fibrous tissue. With the ingrowth of connective tissue blood-vessels penetrate into the vegetation, entering it from the subendocardial layers of myocardium just as they enter sclerotic patches in arteriosclerosis (Koester, v. Langer, Darier, Ribbert), Fig. 172. As healing becomes complete the endothelial layer of the intima slowly grows in from the periphery and gradually covers the entire vegeta-

tion. This relining of the vegetation with endothelium is, from a prognostic stand-point, a most important step in the healing, for, as Wyssokowitch has shown, infection occurs most readily when the surface of the valve is injured, and clinical experience shows that a valve once injured is particularly liable to reinfection. Thus, it is common to find a fresh ulcerative endocarditis occurring upon a valve which is already the subject of a chronic endocarditis, several different stages appearing upon the same specimen.

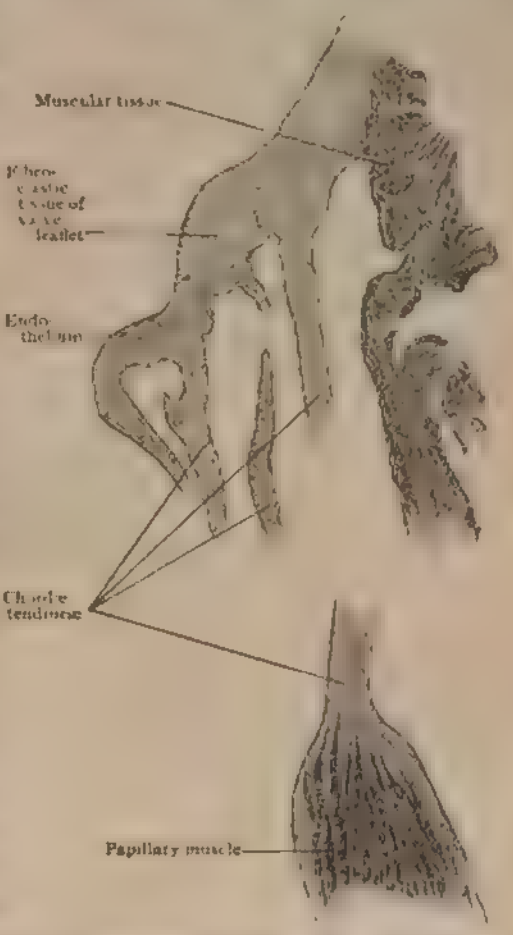

FIG. 173.—Structure of the normal auriculoventricular valve. (After Piersol.)

INFECTIVE AGENTS.

The most important infective agents in the causation of endocarditis are the micrococcus of rheumatic fever, the **pyogenic cocci**, the pneumococcus, the gonococcus, the bacillus influenzæ, and the spirochæte pallida (triponema pallidum) of syphilis.

Rheumatism.—By far the most frequent cause of heart disease is rheumatism, which gave rise to 62.6 per cent. of Horder's cases of malignant endocarditis, and occurs in about the same percentage in the milder forms. However, the exact causal factor of rheumatism itself is not yet settled. Sahli in 1893 isolated what he thought to be a staphylococcus from joints, endocardium, and the heart's blood of patients dying of acute non-suppurative arthritis, and then stated that he "regarded acute articular rheumatism as an infectious disease due to the action of attenuated pyogenic cocci."[1]

Recently Menzer and Rufus Cole have revived this view, and the latter has produced non-suppurative arthritis and endocarditis in rabbits by the injection of streptococci from various sources, showing also that in the joints these assume the diplococcus arrangement.

Triboulet, Wassermann, Westphal and Maikoff, and Poynton and Paine, however, regard the micrococcus (diplococcus) which they have obtained in cases of the rheumatic

[1] ". . . Auffassung des Gelenk rheumatismus als einer auf der Wirkung abgeschwächter pyogenen Kokkenberuhenden Infektionskrankheit."

cycle as a specific organism or at least a specific strain though Walker has shown that its cultural characteristics are by no means sharply defined. The micrococcus (rheumaticus) of Poynton and Paine assumes the diplococcus form in the joints but becomes a streptococcus in culture media, just as Cole found for many ordinary streptococci. Beattie and Longcope also have isolated what they believe to be the micrococcus of Poynton and Paine from cases of arthritis with endocarditis and have produced both conditions in animals. Poynton has obtained the same germ from the cerebral cortex in simple chorea and from the tonsils. Meakins, on the other hand, has found large foci of streptococci in the tonsils which have been removed from patients having rheumatism, but these germs do not show

FIG. 174.—Photomicrograph of a specimen showing acute and subacute endocarditic lesions upon the mitral valve. A, Entire specimen, low power. B, Outline sketch showing the portions from which C, D and E are taken. C, Margin of the area of acute endocarditis, high power. D, Ulcerating area, showing masses of necrotic tissue and exudate. E, Area where the process is more chronic, showing strands of newly formed fibrous tissue entering the vegetation.

any uniformity which would permit them to be identified with the strain of Poynton and Paine. These points tend to favor the original view of Sahli that rheumatism is not due to a single strain but to a variety of attenuated cocci, and is therefore to be regarded as a clinical group of diseases rather than as a single disease.

The Pyogenic Cocci.—The pyogenic cocci of puerperal fever, abscess, and septicæmia are also very common causes of endocarditis. They are identified with special frequency in the malignant forms, owing to the readiness with which they are then cultivated, but there seems little doubt that less virulent strains are responsible for cases of chronic endocarditis as well.

Pneumococcus. — Wells found that the pneumococcus caused endocarditis in 4 per cent. of his 517 autopsies upon cases dying of pneumonia, and hence the latter disease is a relatively frequent cause of endocarditis. Lenhartz states that the endocarditis often arises as a recrudescence after the fever from the original pneumonia has subsided (13th to 15th day), and that it is often malignant and accompanied by meningitis.

Gonococcus.—The importance of the gonococcus in producing endocarditis as well as rheumatism is growing from year to year.

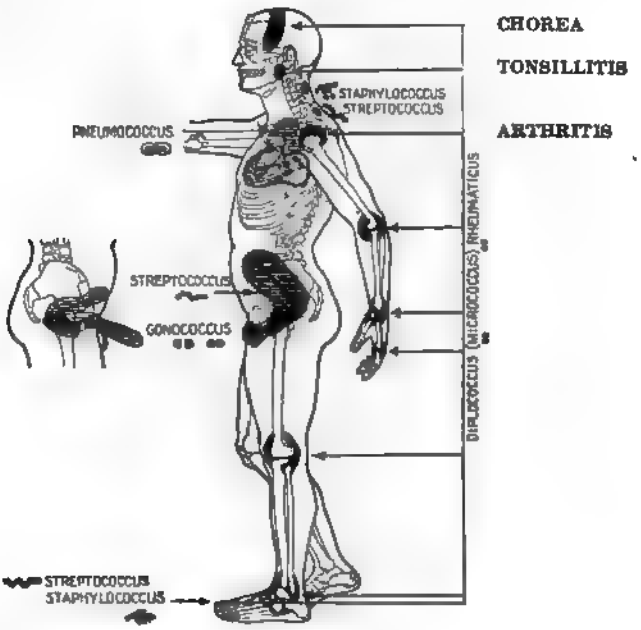

Fig. 175.—Portals of infection in endocarditis. (Schematic.)[1]

The clinical association of endocarditis and urethritis was recognized by Ricord in 1847 and by Brandes in 1854. V. Leyden in 1893 demonstrated upon the valves cocci which decolorized by Gram's method, but the first positive cultures of the gonococcus from the blood during life were made at the Johns Hopkins Hospital by Thayer and Blumer in 1895. Since then the condition has been found frequently, and should always be sought for in cases of gonorrhœal rheumatism.

Miscellaneous Infections.—Occasionally endocarditis arises during or after diphtheria, scarlet fever, and smallpox, though in these cases, as in tuberculosis, the lesion is probably most frequently produced by streptococci which are present as a mixed infection. True tuberculous endocarditis is rare (Marshall), though it has been produced experimentally in animals (Michaelis and Blum).

The bacillus of influenza is also an important factor (Austin), though less frequent in endocarditis than in myocarditis.

[1] The term micrococcus rheumaticus is used for convenience, but with all reservations as to possible specificity.

Syphilis.—Whether true valvular lesions are produced by the spirochæte pallida of syphilis has not been absolutely proved, but recently Collins and Sachs and Longcope have obtained a positive Wassermann reaction in a large percentage of cases of aortic insufficiency in which the valves were puckered, shrunken, and calcified. In these cases it is not the intima but the middle fibro-elastic layer of the valves in which the change goes on, exactly analogous and usually coincident with similar changes in the deeper layers of the intima and media of the aorta.

Sclerotic and Atheromatous Lesions of the Endocardium.—Besides these forms of endocarditis there seems to be a certain number of cases, especially of lesions about the aorta, in which sclerosis and calcification take place in the fibro-elastic layer of the valves exactly as in the luetic lesion, but in which the patient has never had a luetic infection (as in the case of J. L., page 467). The similarity here is exactly like that between luetic and non-luetic arteritis, as shown by Ophuls, and needs no further comment.

PATHOLOGICAL PHYSIOLOGY.

The disturbances in heart action due to endocarditis may depend upon three immediate causes:

(1) The mechanical effects due to leaks or obstructions at any of the valvular orifices. (This will be discussed in detail in connection with each of the chronic valvular lesions.)

(2) The weakening of the heart muscle due to the acute myocarditis and the fatty and parenchymatous changes in the muscle cells, resulting from the direct invasion of the muscle by the cocci, from effect of their toxins upon it, and from the anæmia which frequently accompanies the infection.

(3) The weakening of the heart which, as in other febrile and infectious diseases, results from lowering of vasomotor tone, and which is brought about by a relative emptiness of the blood-vessels. This is accompanied by low blood-pressure and rapid pulse.

In the chronic forms of carditis the first is the most important factor; while in the simple acute and the malignant forms the two latter frequently outweigh it, so that there may be few symptoms referable to the local mechanical effects upon the circulation.

Effects on the Circulation.—The physical signs will be discussed particularly in the case of individual valvular lesions; but in general it may be said that a leak at an orifice *necessitates an increase in the output of the chamber* in order to compensate for the amount regurgitating or an increase in force of contraction of the chamber behind it. Thus, in mitral insufficiency,

Ventricular systolic output = Output into aorta + Backflow into auricle,

while in aortic insufficiency

Ventricular systolic output = Output into aorta = Outflow through peripheral vessels + Backflow into ventricle.

In either of these cases the circulation may be maintained either by increasing this output per beat or by increasing the heart-rate; and in neither of these cases is the pulse-pressure proportional to the systolic output of the ventricle.

On the other hand, when a valvular orifice is narrowed it may have little or no effect until the narrowing reaches a certain point; for, though it slows the inflow or the outflow, as the case may be, yet the duration of systole or of diastole may be sufficiently great to

permit of complete filling or emptying during the time available; but beyond this greater driving power is needed and the chamber behind the stenosis must undergo hypertrophy. Regurgitations usually cause dilatation of the chambers into which the leak occurs, unless a great increase in tonicity of the muscle has caused the cavity actually to decrease in size (Stewart, Cameron, Hirschfelder, Cloetta).

CLINICAL GROUPING.

Clinically, endocarditis (or carditis[1]) has been divided by Osler into three groups:

(1) The malignant type, in which septic and highly febrile symptoms, with symptoms also due to septic embolism in various parts of the body, dominate the clinical picture, and in which the cardiac lesions may spread rapidly and involve almost all the valves. This is usually fatal during the acute attack.

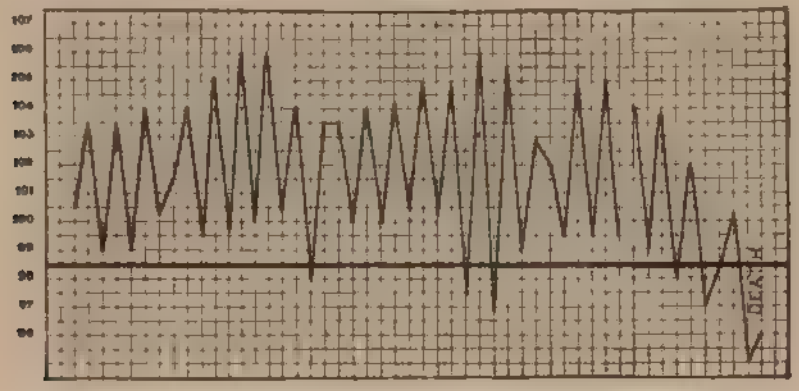

FIG. 176.—Temperature curve from a case of malignant endocarditis.

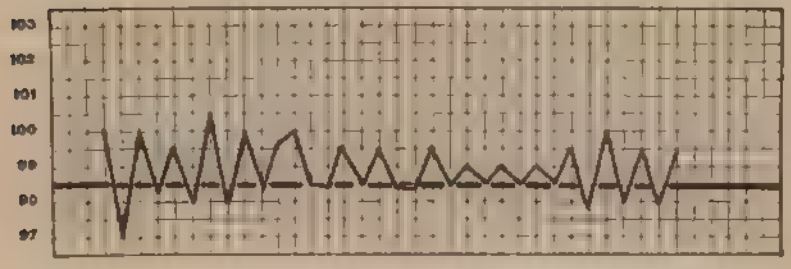

FIG. 177. Temperature curve from a case of simple acute endocarditis.

(2) The simple acute type, in which one or two valves (the mitral and aortic) are affected, but where the lesions remain confined to them. The salient features of the disease are not those due to high fever and embolism, although these may at times be present, but those usually produced by the infective agent; and in addition there are weakness, anorexia, and anæmia, some respiratory distress, and syncope on exertion.

[1] Carditis = inflammation affecting endocardium, myocardium, and pericardium simultaneously.

Occasionally there are œdema, enlargement of the liver, ascites, precordial pain, and palpitation, but these are often absent.

(3) Chronic endocarditis usually follows after an attack of simple acute endocarditis, although it may set in insidiously as a result of progressive sclerotic changes in the valves, especially in association with arteriosclerosis, syphilis, and chronic anæmias. The original characteristics of an infectious disease have subsided, and the picture is entirely due to the mechanical effects of leaking valves and weakened heart muscle; in short, to slowing of the circulation, dyspnœa, cough, œdema, digestive disturbances, palpitation, precordial and referred pains.

MALIGNANT ENDOCARDITIS.

PATHOGENESIS.

Most commonly an attack of malignant carditis is not the first heart disease from which the patient has suffered, but it is found that the acute process involves a valve which already shows marks of a chronic endocarditis. This is not at all surprising, since Rosenbach, Wyssokowitsch, Hasenfeld, and others have shown that valves once injured become the seat of inflammatory processes much more readily than when intact. If the original lesion is a very chronic one and the vegetation well supplied with blood-vessels (Ribbert), organization of the exudate may go on even more readily than in an intact valve; but if the older exudate is still fibrinous or fresh, the tendency to soften and ulcerate is greater than if it were resting upon a base of relatively healthy tissue.

Often the second and malignant infection may be due to an organism quite different from that producing the first, so that one frequently finds a malignant endocarditis, due to the streptococcus, the pneumococcus, or the gonococcus, attacking vegetations originally of rheumatic origin.

In the chronic forms of valvular lesion the symptoms of an acute febrile disease have disappeared, and are replaced by clinical pictures with characteristic forms of hypertrophy, stasis, and murmurs.

Occurrence and Distribution.—The relative rarity of malignant endocarditis is shown by the fact that only 45 cases have occurred among 23,200 admitted to the Johns Hopkins Hospital (0·19 per cent.), as compared with 1781 (7·6 per cent.) of chronic valvular disease; also by the statistics of Horder, who encountered 150 cases among 19,894 patients admitted to St. Bartholomew's Hospital (0·75 per cent.). The occurrence of Horder's 150 cases as regards age was as follows: Under 5, 2 cases; 5 to 10 years, 5; 10–15 years, 9; 15–20 years, 29; 20–30 years, 39; 30–40 years, 31; 40–50 years, 23; 50–60 years, 8; over 60, 4.

The involvement of the valves was: Mitral, 38; aortic, 22; mitral and aortic, 63; tricuspid and mitral, 14; pulmonary and mitral, 7. There were mural auricular lesions in 43, mural ventricular in 8, congenital heart lesions in 8.

Other signs and complications were: Enlarged spleen, 47; heart failure, 66; hæmaturia (sometimes only microscopic), 46; petechiæ, 43 (on legs only in 10); brain symptoms in 22; choreiform movements in 7; retinal hemorrhages noted in 5, though certainly more frequent); embolic aneurisms, 20.

TYPES OF MALIGNANT ENDOCARDITIS.

Osler in his masterly lectures has divided the cases of malignant endocarditis into three clinical groups, which may present both acute and chronic forms.

1. The septicæmic, in which the symptoms are primarily those of septicæmia.
2. The typhoidal type, which closely resembles severe typhoid fever or acute miliary tuberculosis,—continuous high fever, enlarged spleen, and absence of other localized symptoms.
3. The cerebral type, dominated by embolism of the brain, coma, meningitis, paralyses.

Septicæmic Type.—The septicæmic type is the most common and typical, usually following abscess, puerperal fever, operation, wounds, occasionally tonsillitis or quinsy, or some other definite infection, and is characterized by prostration, anorexia, malaise, frequently headaches, and shaking chills. In Horder's 150 cases the fever was continued in 12, irregular and intermittent in 37, quotidian in 40, absent in 5. The temperature sometimes fell for a period before death. The complexion has the sallow yellowish color of hæmatogenous jaundice, there is rapidly progressing increasing anæmia, and the eyes are dull. There is sometimes acute purulent conjunctivitis, sometimes disturbances of vision or even blindness due to the presence of minute emboli or hemorrhages upon the retina. The cheeks are sunken; the skin is usually dry except during the rigor (in contrast to the drenching sweats of rheumatic fever); the tongue is dry and furred; the lungs may be clear or septic bronchopneumonia may be present. Respiration is usually rapid. The signs over the heart are variable. In some cases there are no abnormalities in heart sounds, cardiac area, or in pulsations, except for a rapid pulse-rate, and then the diagnosis may long remain obscure; or, on the other hand, the loudest murmurs may be present both in systole and in diastole, and these have a distribution corresponding to almost any of the valvular lesions, or more usually to several lesions combined. These signs often change markedly from day to day, corresponding to the progression of the lesion from valve to valve, the growth of the individual vegetations, or the disappearance of the latter as they slough off into the blood stream. The pulse is small and collapsing, but usually too rapid for dicrotism, and the blood-pressure is low (maximal 85 to 110 mm., minimal 60 to 90). It becomes larger and more typically water-hammer in character, and the diastolic pressure falls to 40–50 mm. if a leak sets in at the aortic valve. The loud systolic murmur over the tricuspid area, corresponding to tricuspid insufficiency either functional or organic, is among the most common in malignant endocarditis, for this valve bears the brunt of both the increasing organic lesions and the progressive weakening of the heart muscle. Accompanying this there is also systolic pulsation in the jugular vein. A diastolic murmur may be present to either left or right of the sternum, and may correspond to either aortic or pulmonic insufficiency, the distribution in the latter case being somewhat different from the former. A pericardial friction, associated with the onset of fibrinous or purulent pericarditis, is not uncommon.

The liver is frequently enlarged, either from cardiac weakness or from a definite suppurative hepatitis and cholangitis. When associated with tricuspid insufficiency it may pulsate with systole.

The spleen is often enlarged, from the presence of infarctions of greater or less extent. The abdomen may be otherwise normal or may be tense, and there may be local tenderness and muscle spasm from

localized infection or general peritonitis; not infrequently these areas correspond to the uterus (especially in puerperal endocarditis) or to the kidney, owing to infarction, in which case there are also albuminuria and hæmaturia.

There is sometimes oedema of the extremities. Arthritis is frequent, often accompanied by injury to the epiphyses as well as the joints, and occasionally by spontaneous fractures. The skin may show very numerous small purple petechiae or large areas of ecchymosis; or, on the other hand, there may be numerous subcutaneous abscesses of varying size.

The blood count is usually low, especially the hæmoglobin, corresponding to the type of a secondary anæmia. Sometimes 500,000 erythrocytes are destroyed each day. There is almost always a polymorphonuclear leucocytosis (20,000-30,000). As has been seen blood cultures are positive in about 90 per cent. of the cases,—colonies of the infective agent, usually 20-40 per cubic centimetre of blood.

The urine is of variable amount, and specific gravity usually high. It generally contains albumen and casts, and often there is definite hæmaturia due to infarction of the kidney. Sometimes the blood can be seen only with the microscope. Not infrequently there is cystitis with cocci in the urine in considerable quantities.

Typhoidal Type.—The cases of the typhoidal type are characterized also by asthenia, by high fever (103° to 106°), which is more or less continuous, frequently flushed face, dry tongue, sometimes coma vigil and picking at bedclothes, enlarged spleen, but otherwise no definite localizing symptoms. There may be a slight bronchitis or small foci of bronchopneumonia. The cardiac signs may be indefinite, or may be thought to be remnants of old valvular lesions. The differentiation from typhoid fever on the one hand and acute miliary tuberculosis on the other may be impossible by the simple methods of physical diagnosis, and the diagnosis must rest with the blood culture. Occasionally the presence of petechiae in the skin may suggest typhus fever. Examination of the eye-grounds may show small white spots of retinal exudation and occasional hemorrhages, but the picture may be difficult to distinguish from typhoid lymphomata or miliary tubercles. The presence of leucocytosis is suggestive, but not decisive, while the absence of Widal reaction is of value only as negative evidence. The only decisive evidence is given by the blood culture.

Cerebral Type.—In the third or cerebral type the symptoms due to embolism of the brain and usually of the left middle cerebral artery dominate the picture. There is a history of onset with fever, weakness, and perhaps chills, perhaps a shower of petechiae over the body, and hæmaturia, and then a sudden onset of hemiplegia, with, or more usually without, convulsions, and perhaps relapse into unconsciousness. The patient is then left with unconsciousness, hemiplegia (usually right-sided), and usually aphasia, more or less disturbance of vision, and choked disk. The septic infarct may also give rise to purulent meningitis so that there may be unconsciousness, rigidity of the neck, and Kernig's sign as well, and the cerebrospinal fluid obtained from lumbar puncture may be under high tension, cloudy, rich in albumen and in cocci.

These lesions are produced by septic emboli of varying sizes carried off the necrotic surfaces of the infected valves. In Horder's series they occurred in 14.6 per cent. of the cases. The symptoms vary in character and severity, according to the location and extent of the lesion, from a few choreiform movements to paralyses, convulsions, and coma. Aphasia is, of course, relatively common.

The diagnosis of the primary condition may depend upon the varying heart signs and the positive blood culture.

CHRONIC INFECTIVE ENDOCARDITIS (OSLER).

Osler has called attention to the existence of a chronic form of malignant endocarditis, which may last from four to fourteen months. It is characterized by an asthenic condition, with remittent or intermittent fever rising to a maximum of 102°–103°, chills and sweats, in about 60 per cent. of the cases, petechiæ, especially upon the shins, enlarged spleen, and heart signs, which vary as the process extends from valve to valve, or the valve substance sloughs. There is usually a progressive anæmia. The leucocytes are almost always increased (10,000 to 15,000 per c.mm.), though not so greatly as in the acute forms, and the blood culture is usually positive. However, in this condition more than in any other, it may occur that a single blood culture may be negative, whereas a subsequent attempt may give a good growth, for the germs are apt to pass into the blood in showers.

In Osler's experience the disease was always fatal, but Horder reports one case with recovery.

DIAGNOSIS.

The diagnosis of malignant endocarditis often presents considerable difficulty. The differentiation from pneumonia may be especially difficult, since there are usually small areas of septic bronchopneumonia present. On the other hand, as Rosenow has shown, the blood culture in pneumonia often yields large numbers of pneumococci, and this germ is not infrequently the cause of malignant endocarditis.

Thompson has also called attention to the fact that acute hyperthyroidism (Basedow's disease, exophthalmic goitre) may present a clinical picture of fever, chills, sweats, tachycardia, dilated heart with systolic murmurs, which closely simulates that of malignant endocarditis. The thyroid in these cases is enlarged and tender and the ocular signs are usually pronounced.

The crucial points in the differential diagnosis of malignant endocarditis are, therefore, given in the following table:

From pneumonia—petechiæ, signs of valvular lesions.
From typhoid fever—by leucocytosis, absence of Widal reaction, blood culture.
From rheumatic fever—by enlarged spleen, petechiæ, chills, blood culture.
From malaria—by absence of plasmodia, leucocytosis, heart signs, positive blood culture.
From miliary tuberculosis—by leucocytosis, heart signs, absence of tubercle bacilli, positive blood culture.
From cerebrospinal meningitis—by absence of intracellular diplococci in cerebrospinal fluid, positive blood culture yielding other germs.
From acute Basedow's disease—by positive blood culture, absence of oculomotor signs of Basedow's disease, polymorphonuclear leucocytosis.

CASE OF MALIGNANT ENDOCARDITIS.

Margaret P., aged 12, factory worker. Previously healthy except for scarlatina at 5 years and measles at 7. Never had rheumatism or chorea. Two weeks before admission she had a shaking chill, followed by fever and sweats. She has felt weak, nauseated, has vomited every day, and is irrational on the day of her admission. Has had no headache nor epistaxis.

At the time of her examination by Dr Cole she was irrational, chilly, and shivering. Her color was a dusky pallor with slight cyanosis. Slight enlargement of glands; lungs clear except for a few mucous râles.

Heart.—Apex impulse cannot be seen or felt. Dulness extends 7 cm. to the left and 2 cm. to the right of the midline, and above to the second rib. At the apex there is a well-marked systolic murmur, transmitted as far as the anterior axillary line. Pulse is regular, of fair volume, 105 per minute.

The border of spleen is just felt. Liver dulness extends to the costal margin. Reflexes: Knee-jerks active, no Kernig's sign. Rectal examination negative. Vaginal smear shows no intracellular diplococci. Blood count, Nov. 4. Red blood-corpuscles 4,352,000. Haemoglobin 80 per cent. Leucocytes 31,460.

The maximal blood-pressure ranged between 80 and 105 mm. Hg; the pulse-rate between 120 and 210. Differential count showed polymorphonuclear 96·4 per cent., large mononucleurs 1.2 per cent., small lymphocytes 2·2 per cent. No malaria parasites in the blood. Widal and blood cultures persistently negative.

The patient's general condition remained about the same. On Nov. 6 a few ecchymoses appeared on the back and abdomen. By Nov. 7 the systolic murmur was well heard in the axilla. Lumbar puncture gave a clear sterile fluid under pressure of 280 mm. (slightly elevated). Nov. 12. A few pin-point vesicles appeared on the abdomen, along with new petechiae on abdomen and face. Nov. 14. Patient better and temperature lower. Nov. 22. Had a severe shaking chill; slight epistaxis. Nov. 26. Felt faint while in the tub. Nov. 28. Haemoglobin 65 per cent. Nov. 29. Cardiac dulness has increased, extending 9.5 cm. to the left and 4 cm. to the right; above to the second left interspace. The murmur is about as before, the pulse large and collapsing. Dec. 1. Anaemia has increased. Red blood-corpuscles 3,800,000; haemoglobin 60 per cent.; leucocytes 27,000. Dec. 4. A well-marked presystolic thrill is felt at the apex. In the afternoon she complained of pain in the feet and loss of sensation in feet and legs. She cannot feel touch below the knees. The feet are warm, no discoloration, knee-jerks are present. Dec. 5. Red blood-corpuscles 2,300,000; haemoglobin 50 per cent.; leucocytes 31,000. The patient's condition became worse and crops of petechiae appeared. The pulse became irregular in force and rhythm. She died on Dec. 16.

Autopsy by Dr MacCallum showed acute vegetative endocarditis of the mitral valve, cardiac hypertrophy, œdema of the lungs, acute splenic tumor with anaemic infarction, acute diffuse nephritis with anaemic infarction, embolic occlusion of the aorta at its bifurcation. A motile coccus (micrococcus rubescens) was found in the heart, micrococcus albus and bacillus pseudodiphtheriæ in the vegetations, and in the kidney an unidentified actinomyces, sarcina flava, and micrococcus albus.

TREATMENT.

The treatment of malignant endocarditis is the treatment of any form of general septicæmia, absolute rest, very light, soft or milk diet amounting to as near 3000 calories per day as possible, and avoidance of excitement. Drugs are of little value. Strychnine may be given in doses of 2–3 mg. ($\frac{1}{30}$ to $\frac{1}{20}$ gr.) every four hours, or digitalis also, with a view of increasing the activity of the vasomotor centre and the tonicity of the heart; but little is accomplished by their use, and in some cases the heart muscle is already so much injured by the infection that further stimulation is actually harmful. Salt infusions may be given, but they serve to

swell the volume of blood, to dilate the heart, and to increase its work, and, although they may perhaps "wash out the toxic substances through the kidneys," it is doubtful whether they are at all effectual.

Intravenous Injections.—Intravenous infusions of collargol and other metallic compounds have been tried and some apparently favorable results reported, but these have invariably been shown to be overestimated when the work was repeated by more careful observers. The antistreptococcus serum of Marmorek has been used in cases of malignant streptococcic endocarditis, but this also has no value.

Inoculations with Bacterial Vaccines.—More recently A. E. Wright has instituted the method of inoculating the patient with small doses of killed cultures of the germ causing the infection in the hope of thereby increasing the production of protective substances. Though this is the most promising of all the methods, it has failed to give satisfactory results in the hands of careful observers such as Rosenow and Horder.

SIMPLE ACUTE ENDOCARDITIS.

The malignant forms of carditis described in the foregoing chapter are relatively infrequent (0.19 per cent. of admissions to the Johns Hopkins Hospital). Much more common are the milder infections which assume the form of simple acute or subacute endocarditis, and in which the symptoms are often referable mainly to a mild subacute fever and anæmia, and with comparatively less frequency stamped with the typical features of heart disease, so that the latter may become evident only on physical examination.

Rheumatism.—Like the malignant form, which is usually of pyogenic origin, the simple endocarditis is far more frequently rheumatic (Bouillaud, 1835), manifesting itself in association with other manifestations of the "rheumatic cycle,"—tonsillitis, rheumatic arthritis, chorea, pleurisy, or the rheumatic erythemata; rheumatism being the etiological factor in 65.6 per cent. of all cases of endocarditis in the Medical Clinic at Zurich, in 36.7 per cent. at Jena, and in 58 per cent. at Leipzig.

Similar figures appear from the clinics of Great Britain and America, the statistics of the Johns Hopkins Hospital being quite according to the rule.

A much higher percentage of the cases of rheumatism acquire endocarditis than is true of any other disease. It was present in 61.3 per cent. of all cases of this disease in children in West's series, in 66 per cent. of Fuller's cases and in 80 per cent. of the cases reported by Cadet de Gassicourt.

Gibson states that the likelihood of endocardial infection is proportional to the severity of the rheumatic affection.

The same relative frequency applies also to chorea, the other important member of the rheumatic cycle. Stephen Mackenzie finds 60.6 per cent., Donkin 10 per cent., Osler 51.4 per cent. affected with carditis, although the arthritic history is often absent.

Other Infections.—Other diseases, though occasional causes, are much less frequently followed by endocarditis. Thus, Osler found it twelve times in 216 autopsies upon cases of phthisis, five times in 100 pneumonia cases, twice in 80 autopsies upon typhoid fever, and he states that it is not uncommon in scarlet fever. In most of these cases the secondary infection with streptococcus is probably responsible for the condition. Influenza, smallpox, measles, and diphtheria also are occasional etiological factors. In all these diseases any overwork or other overstrain upon the heart during the course of the infection increases its susceptibility and enhances the liability to affection of the endocardium just as, according to Poynton, fright (or hard study at school) predisposes to affection of the brain, namely chorea.

Age.—As to age it may be said, that, in contrast to both the malignant and the chronic forms of endocarditis, the simple acute carditis which represents the usual beginning of the process presents itself most frequently in children, especially in the second decade of life, and that the age of maximum frequency is the age of the greatest exposure, the second and third decades.

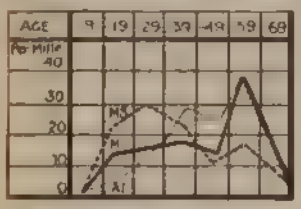

FIG. 178.—Diagram showing relative frequency of the most important valvular lesions at various ages. Modified from Osler. Solid line = mitral insufficiency, broken line = mitral stenosis, dotted line = aortic insufficiency. The figures indicate the decades. Under 9 years, 9, 10–19, 19–29, 29–39, etc.

The frequency is due merely to the fact that rheumatism is then more frequent, although when contracted in childhood it appears to be followed by a greater percentage of cardiac complications than in older persons, and pericarditis (especially adherent pericardium) and myocarditis are more severe. Of 145 cases under 15 years of age Holt and Crandall found under 5 years 14 cases, 5–10 years 71 cases, and 10–14 years 60 cases, 38 per cent. being males and 52 per cent. females. It is especially noteworthy that of these 145 cases almost 90 per cent. were brought about by diseases of the rheumatic cycle, in contrast to 60–65 per cent. in older persons. Indeed, the earlier in life the rheumatic infection is contracted, the more it assumes the type of a general carditis and the less severely are the joints involved. As many writers have stated, rheumatic fever in children usually assumes the form of a tonsillitis, with carditis and chorea, and is frequently devoid of any arthritic symptoms whatever.

The myocarditis has been discussed in Part II, Chapter IX and is an important feature. The weakness of the heart muscle which results leads to dilatation and overfilling of the chambers, and this in turn increases the leaks due to the lesions upon the valves.

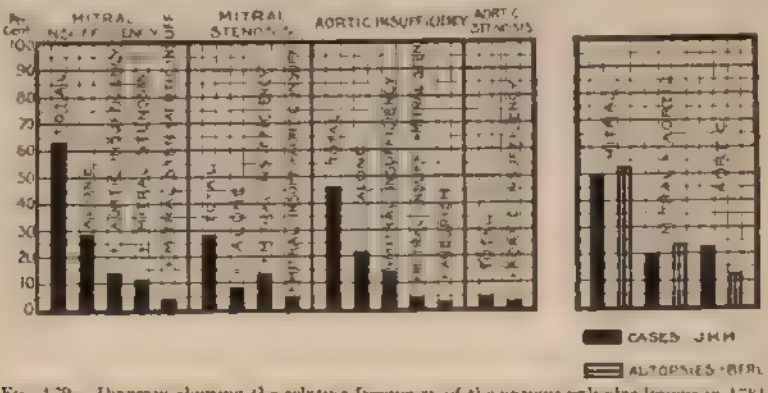

FIG. 179.—Diagram showing the relative frequency of the various valvular lesions in 1781 cases of valvular heart disease admitted to the Medical Service of the Johns Hopkins Hospital from 1889 to 1908 as well as those found by Sperling in 300 autopsies in Berlin. Small space, 5 per cent. Berl., Berlin. J. H. H., Johns Hopkins Hospital.

Sex. Many authors, among them Osler, state that men are more frequently affected than women, though v. Jurgensen states that valvular disease occurs with practically the same frequency in both sexes.

INVOLVEMENT OF INDIVIDUAL VALVES

The relative frequency with which the valves are involved is shown by the analysis of 1781 cases of endocarditis admitted in the Johns Hopkins Hospital from 1889 to 1908, represented diagrammatically in Fig. 179. The figures show a general correspondence to those of v. Jurgensen in 2170 cases in the German clinics.

PATHOLOGICAL PHYSIOLOGY.

The pathological physiology of simple acute endocarditis presents the condition due to the individual valvular lesion (to be considered in detail in the appropriate chapters dealing with the chronic endocarditis), modified or added to by an element of diminished vasomotor tone due to the acute febrile condition. As the result of this vasodilatation, especially in the abdominal area, the blood collects in the dilated veins and capillaries, the blood-pressure may be low, and the symptom complex of arterial anæmia or low blood-pressure sets in. Moreover, there is usually a certain degree of actual anæmia added to the lesion, and this often increases the difficulty in breathing; although it does not, as a rule, bring on the red and purple hue of chronic cyanosis. Still further the increase in the leakage causes damming back and secondary dilatation of the chambers behind it, œdema in the walls of the ventricles and in the valve cusps, and increased susceptibility to infection.

PATHOLOGICAL ANATOMY.

The endocarditis itself is less severe than in the malignant form. Fewer bacteria are deposited upon the valves, and these show less tendency to multiply, so that the process of organization, as a rule, outraces necrosis, and consequently the separation of emboli is rare. The valves thus show an injured surface covered by a more or less thick or exuberant layer of fibrin, with active organization proceeding upward from its base. This may be seen in any stage of advancement, from fresh fibrin in the early stage to completely organized firm young connective tissue, covered by intact endothelium, when healing has become complete.

Pathologically, the difference between the malignant and the simple endocarditis is merely the usual difference between a mild and a virulent infection of any tissue. There may be no actual difference in etiology, and the malignant form may represent only a very virulent strain of the same organism which would ordinarily produce a milder infection; or, on the other hand, micro-organisms of the same virulence may produce different types of lesion in persons with different powers of resistance.

SYMPTOMS.

It is particularly noticeable that in these cases during the first attack the symptoms due to distinct heart failure are largely absent, and the main symptoms are those of accompanying rheumatic disease, along with the weakness, pallor, and anæmia (usually about 60 per cent. hæmoglobin), such as might be due to any mild fever, though occasionally, as in the case of J. A. (page 316), the onset of aortic insufficiency is attended by pain and sudden collapse. The temperature rarely attains 101° unless an acute arthritis or acute pneumonia is present. The pulse is usually rapid and regular. Its quality depends upon the nature of the lesion, being large and collapsing in the presence of aortic insufficiency, small in mitral stenosis, and of moderate size in mitral insufficiency. The blood-pressure is sometimes above, sometimes below normal.

PHYSICAL SIGNS.

As a rule, the patient does not seem very ill, he has sometimes an anxious expression, is usually pale and sallow, in contrast to the older cases of mitral disease, who usually show a flushed and cyanotic hue. Occasionally choreic movements are present. It is very common for the tonsils to be enlarged, since these are the usual portals of entry for the rheumatic infection, and there is frequently a yellow exudate in the crypts or a membrane over their surfaces. In almost all rheumatic cases there are foci of cocci (streptococcus or micrococcus rheumaticus?) in the deeper tissue of the tonsil. Along with this infection the so-called tonsillar lymph gland just below the angle of the jaw and often the submaxillary and anterior cervical lymph glands are enlarged. The chest shows no special peculiarity except that precordial bulging is often present, especially marked in children (see page 88), even in the first attack of endocarditis. The cardiac signs are the same as for the chronic valvular lesions, though usually less marked. They will be discussed in detail under the special chapters.

The liver is usually not enlarged unless there is marked heart failure. Occasionally the spleen is palpable and even hard, tender, and painful, as a result of a fresh or old infarct, and this condition may persist unchanged for years.

A few months ago the writer saw in the Johns Hopkins Dispensary a young girl in whom a large, very hard spleen had been present for several years, first appearing during a rather severe attack of simple acute mitral endocarditis.

There is often slight oedema of feet and ankles, though very many cases come to treatment before this has set in. The presence of œdema in an early acute endocarditis is a rather grave sign, since it indicates the failure of the heart to respond promptly to the added load.

The urine is usually of high specific gravity and contains a small amount of albumen and a few coarsely or finely granular casts,—a typical febrile albuminuria.

The blood examination usually shows a slight grade of secondary anæmia

SUBSEQUENT COURSE.

As in the cases cited on page 316, there is usually gradual improvement under any treatment in which the main factor is sufficient rest, during which the infection subsides (the bacteria dying, or more commonly becoming latent), the vegetations undergo gradual organization and more or less thickening or shrinkage, and fever passes off, as does the acute myocardial weakness. The patients almost always recover from the first attack. Recurrence is especially common, and is the danger against which especial precaution must be taken, the more so as the second attack often spreads to another valve or even to two more. It is the liability to repeated attacks which keeps the pathological process ever fresh and increasing. There is then usually a little area of incompletely organized fibrin always present to give soil to any stray micrococcus that may be carried by the blood stream, and thus produce a new outbreak of fresh endocarditis with exacerbation and perpetuation of the old symptoms. After a single attack,

especially when one only is involved, complete organization of the vegetation may set in, the acute myocardial changes subside, and the heart muscle may soon regain its normal function.

Compensation.—A slight leak (see page 322) may remain at the site of the vegetation, just enough to produce a murmur and perhaps even bring about slight hypertrophy, but without really impairing the function of the heart; and the individual who suffers from no further acute endocardial changes may go on for thirty or forty years, until the age of sclerosis sets in and the leak is widened by sclerotic shrinkage, without the appearance of any further symptoms. On the other hand, as da Costa has shown, persons with old perfectly compensated valvular lesions are much more susceptible to cardiac overstrain and acute dilatation than are normal individuals. With the dilatation there comes a functional insufficiency of the valves, which adds its effect to that of the organic lesion; and finally, as Roy and Adami have shown, stasis brings about œdema and cellular infiltration in the cusps. This infiltration is followed by further valvular sclerosis and shrinking, and thus the cardiac overstrain in itself tends to increase permanently the original lesion

When hypertrophy and compensation are good and the individual either lives a quiet life or has developed his muscles gradually to meet the strain of his surroundings, he may escape overstrains entirely, and the lesion may either be stationary or may shrink by gradual sclerosis. It is a rather common occurrence to find perfectly healthy young adults or even active men in middle age who have had well-compensated mitral insufficiency present since childhood. The same is also true of aortic insufficiency except that this usually again makes itself felt about the age of arteriosclerosis, *i.e.* about the age of forty. Even then, with good care, general hygiene, avoidance of muscular overstrain, nervous excitement, and overeating, great moderation in the use of alcohol and tobacco, and especially personal prophylactic measures against infectious diseases, a long life may be attained by the patient.

Reinfection.—On the other hand, when the patient is still subject to recurrence of his rheumatism or tonsilitis, or to repeated attacks of pneumonia, bronchitis, or influenza, the probability that the cardiac lesion will remain quiescent is a small one, and it becomes more likely that both valve and muscle will suffer further changes whose limit it is impossible to predict. It is therefore most important not to give a definite prognosis to the family or friends of the patient until he has been under observation for about a year after the attack of endocarditis has subsided, so that all these factors may be carefully watched and taken into account, prophylactic measures be instituted, and the recuperative power of the heart muscle be gauged.

Complications.—Another factor even more important than the endocardial lesion is the *involvement of the pericardium* and especially the production of *adherent pericardium*, so common in the first and second decades. This condition perhaps more than any other leads to early heart failure, since it imposes the greatest strain of all upon the heart, and, as it develops insidiously and frequently reaches its maximum only after the first acute attack has passed off, it should be watched for with great care.

SIMPLE ACUTE ENDOCARDITIS.

J. A., male, cannery worker, aged 15, entered the hospital complaining of rheumatism. He has been a rather delicate boy, having had erysipelas, measles, whooping-cough, and chicken-pox when a child, and attacks of definite articular rheumatism at nine and ten years. He has done soldering in a cannery for the past two years.

About five weeks before admission he began to complain of pain in his ankles and knees, for which he was put to bed. At this time his physician found a temperature of 104°, and he had chilly sensations, but no shaking chills. About two weeks later while lying down he felt an intense pain in his heart and began to get white in the face and blue at the lips. Since then, though he has been losing weight and strength, he has had no more pain. He has had occasional headaches during the illness.

Examination shows a well-nourished boy of sallow color, with injected pharynx, enlarged tonsils, and enlarged posterior cervical and axillary lymph-glands. Chest is well formed and lungs are negative but for a few moist râles over the left apex.

Heart. There is marked precordial bulging. The apex beat is seen in the 4th left interspace 9 cm. from the midline. Dulness extends 4 cm. to the right of the midline and above to the second rib. There are no thrills. The first sound at the apex is preceded by a short rumble (Flint murmur) and replaced by a soft blowing systolic murmur. The second sound is clear at the apex, but at and near the sternum is followed by a blowing diastolic murmur, maximum over the insertion of the third right rib. The pulse is 124 per minute, small but definitely collapsing, and there are well-marked capillary pulsation and throbbing of the carotids. Blood-pressure maximal 115–125 mm. Hg.

Joints. There are swelling of right elbow and left ankle and soreness of elbows, knees, and right hip, slight wasting of interossei of hands and feet. Genitalia and reflexes are normal.

There is no œdema. Red blood-corpuscles 5,000,000, hæmoglobin 75 per cent.; leucocytes 11,000. Urine.—Lemon yellow. Specific gravity 1015; alkaline; no sugar, a trace of albumen; a considerable number of coarsely granular casts.

Oct. 31. Dulness extends 7.5 cm. to the left of the midline and 2.5 cm. to the right. Nov. 13. Red blood-corpuscles 5,400,000; hæmoglobin 80 per cent.; leucocytes 6,600. General condition is excellent. Pulse continues rapid. The joints are clear. Jan. 5. There has been gradual progressive improvement. Red blood-corpuscles 4,700,000; hæmoglobin 90 per cent.; leucocytes 11,000. There has been a gradual rise in the maximal pressure to 150–160 mm. Hg, as the patient's improvement has continued in spite of the rapid pulse. The patient was discharged quite well on Jan. 17, but had a second more severe attack several years later.

TREATMENT.

The treatment of the acute attack of endocarditis partakes in general of the treatment of a mild febrile disease or a secondary anæmia on the one hand, and of the particular valvular disease on the other. Rest in bed until a couple of weeks after the subsidence of all febrile symptoms is therefore an absolute necessity, also light and easily digestible diet, at first of 800–1000 calories, later 2500.

Digitalis and Strychnine.- As a rule, digitalis is not absolutely necessary, and is dispensed with by most Anglo-American practitioners.

However, Cloetta has shown that the hearts of animals in which aortic insufficiency has been produced experimentally recover much better, undergo much less dilatation and acquire much greater strength if digitalis treatment is begun at once and is continued over long periods (about a year), than if this treatment is omitted. Cloetta claims equally good results in man, but his cases are too few to warrant conclusions. Nevertheless, the results are sufficiently definite to warrant the prolonged use of digitalis in small doses (0.3 to 0.6 c.c., ℥v to x of the tincture) in cases of acute endocarditis with cardiac dilatation.

In cases in which digitalis is not used strychnine should be given in doses of 2 to 3 mg. ($\frac{1}{30}$ to $\frac{1}{20}$ gr.) three or four times a day.

The salicylate preparations, sodium salicylate, salol, salipyrin, aspirin, etc., should be given for the rheumatism; but, although they certainly relieve the pain, and it has been shown that they are excreted into the joint cavity, the duration of the fever and arthritis does not seem to be much affected by them, and certainly the frequency of cardiac involvement is unchanged. On the other hand, the salicylates, especially in large doses, have a depressant effect upon the heart, and the use of these drugs should therefore be as restricted as is consistent with relief of arthritic pain.

According to many authorities, the salicylates seem to be more effective when injected directly into the joint or into the tissues immediately surrounding it. The writer's experience with this method is limited and in the cases tried its results were not striking, but it is sometimes worthy of trial. Oil of wintergreen (Oleum gaultheriæ, methyl salicylate) applied to the skin over the joint also seems to cause great relief of pain, but it is possible that the rubbing may also cause more of the micrococci to be thrown out in the blood stream than might otherwise be the case. Hot compresses of saturated aqueous solutions of oil of wintergreen to the joint may suffice to allay pain.

Other Therapeutic Measures.—It is most important to relieve anæmia, which is usually present and which is always a contributing factor to the fatty degeneration and weakness of the myocardium. Rest, full diet especially rich in eggs, milk, and green vegetables, and administration of iron usually relieve this symptom.

The iron may be administered as Pil. ferri carbonatis (Blaud's pills), 0.2 to 0.3 G. (gr. iii to gr. v) t.i.d., p.c.; or Massa ferri carbonatis (Vallet's mass, a more stable preparation containing honey instead of sugar); Elixir ferri, quininæ et strychninæ, 8 c.c. (℥ii) t.i.d., a.c.; or as Syrup. ferri iodid, 1 c.c. (♏ xv) t.i.d., p.c.

If the anæmia is severe or does not yield to iron alone, arsenic should be given as well, since it has been shown that iron and arsenic together accelerate formation of red corpuscles and hæmoglobin more than does either drug alone.

Arsenic is usually given in the form of Liquor potassii arsenitis (Fowler's solution), beginning in doses of 0.2 c.c. (♏ iii) t i.d., p.c., and increasing one drop at each dose until 1 c.c. (♏ xv) is reached or puffy eyelids and albuminous urine show that the physiological limit has been reached.

Prophylactic Treatment.—One of the most important factors in hastening the healing of a fresh vegetation is to keep it from being reinfected by bacteria floating in the blood stream. Every focus of infection is a storehouse from which a few bacteria are given off from time to time, and hence is a source of danger. Accordingly in a number of clinics, and particularly in the medical clinic of the Johns Hopkins Hospital, under Prof. Barker's direction, an effort is being made to stamp out every focus of infection to be found anywhere in the body. Carious teeth, paronychias, and ischiorectal abscesses are removed. Particular attention is given to the tonsils. These organs are the main portals of entry for the rheumatic infection. In persons who are subject to recurrent tonsillitis there are almost always small abscesses containing cocci persisting in the depths of the tonsillar tissue, even when there is no inflammation visible upon the surface. These are perma-

nent portals of infection. Dr. Barker therefore insists upon the removal of enlarged tonsils in most cases of rheumatic heart disease. This should be done between but not during the attacks, since there is danger of throwing more cocci into the blood. The improvement which follows removal is sometimes immediate and striking. The patient's color improves within a few days. He feels better. His expression is brighter, and he appears more robust. Improvement is more rapid and, since reinfection is less frequent, it is more permanent.

It is naturally of great importance that all the tonsillar tissue should be removed, since a small amount left in place may again undergo hypertrophy and become reinfected. Such complete removal is impossible with the guillotine, the snare, and the electro-cautery, and is extremely difficult by even the ordinary intracapsular dissection. The most satisfactory method known to the writer is the extracapsular dissection as performed by Bordley.

Palliative Treatment of the Tonsils.—By way of palliative or prophylactic treatment various antiseptic gargles may be used. Gargles which contain hydrogen peroxide are to be preferred, because the pus-cells contain a catalase which sets free the oxygen. The nascent oxygen is a powerful antiseptic, and the excess collects in bubbles which mechanically loosen and sweep off the exudate. The hydrogen peroxide should not be stronger than 2 volume per cent. (one part commercial hydrogen peroxide to four parts of water).

Other gargles that may be used are Dobell's solution, dilute Lugol's solution, and dilute potassium chlorate solution (especially with equal parts of dilute hydrogen peroxide).

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II.

MITRAL INSUFFICIENCY.

OCCURRENCE.

Of all the valvular lesions those involving the mitral valve are the most common, especially those which lead to the production of a leak at that orifice (mitral insufficiency, mitral regurgitation, incompetency of the mitral valve).

Involvement of the mitral valve alone was found to be present in 51 per cent. of 1781 cases of valvular disease admitted to the Johns Hopkins Hospital between 1889 and 1908, and in 54 per cent of Sperling's 300 autopsies on similar cases in Virchow's Pathological Institute in Berlin. (Fig. 179.) Mitral insufficiency, both alone and in association with other lesions, was present in 64 per cent. of the Johns Hopkins cases, occurring alone in 29 per cent. (see the Table, Fig. 179).

As regards age, Gillespie (Fig. 178) has found from a study of 816 cases that its frequency is about uniform between ten and fifty years, after which it diminishes. This is in sharp contrast to the cases of mitral stenosis, which are most frequent before the age of thirty and become much rarer after thirty. In youth women are slightly more often affected; in old age the affection is a little more common among men. The mortality from mitral insufficiency becomes greater as age progresses.

PATHOLOGICAL ANATOMY.

Pathologically, cases of mitral insufficiency may be divided into two groups:

1. Organic, due to vegetations, cicatrizations, or atheromatous plaques, thickening of the edges of the valves, or ulceration upon the valve itself.

2. Functional (or relative), in which the valves are intact, but closure becomes imperfect through relaxation of the muscle into which the cusps are inserted, or through stretching of the chordæ tendineæ.

Organic Mitral Insufficiency. — The pathogenesis of organic insufficiency is simple. The lesions arise during the course of an acute or subacute endocarditis, and frequently result from the accumulation of inflammatory exudates from several successive infections. These are cemented into permanent structures by organization and calcification. Occasionally a perforation of the valve occurs from ulceration. As in acute endocarditis, infection in the rheumatic cycle is the most common cause of chronic mitral disease, though other infections may represent not only primary but exacerbating factors.

The vegetation, once formed, gives rise to the leak by holding apart the neighboring portions of the cusps so that regurgitant streams occur about its serrations (Fig. 180, a, A).

Tests for Sufficiency of Mitral Valve. It is easy to demonstrate by the method of Gaul (see page 10) and Meigs that when the vegetation is not extensive the mitral cusps may adapt themselves perfectly to its contour and prevent a leak altogether, but when, as is usually the case, their flexibility is altered by a line of vegetation, atheroma, or infiltration, this apposition is prevented. The amount of blood actually regurgitating, and hence the functional importance of the lesion, depends largely upon these factors, as well as upon the concomitant affection of the cardiac muscle.

At the autopsy table the mitral valves may be tested for leakage in either of the following ways:

(1) Water may be forced into the ventricle through a cannula attached to the water faucet. The valve usually holds against leakage. (T. W. King, 1837; G. A. Gibson, Meigs.)

(2) A slit is made in the ventricle wall near the apex. The heart is turned upside down, the slit at the apex is held open with the fingers and water poured in from above. The normal valve would show no leakage (Bleichroeder).

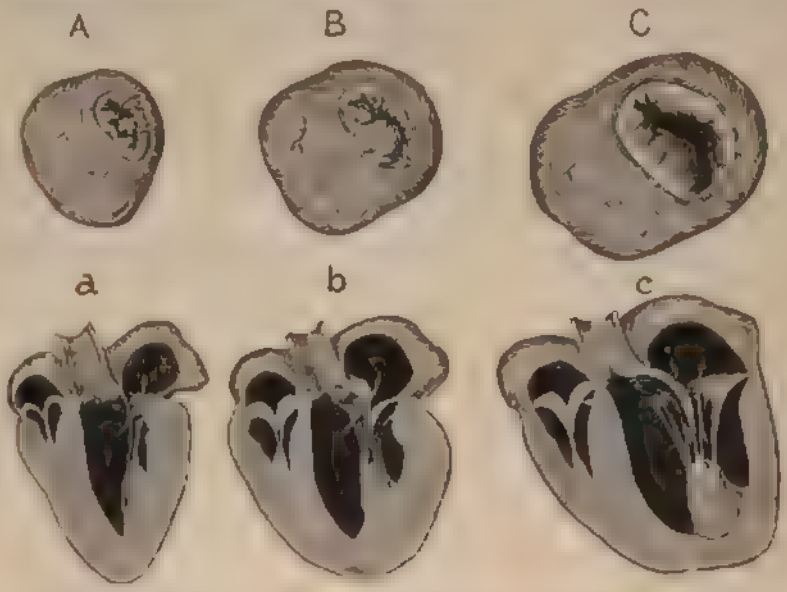

FIG. 180.—Regurgitant streams in organic and functional mitral insufficiencies. A, organic mitral insufficiency. B, functional insufficiency of the papillary type. C, relative mitral insufficiency. a, view from above the valves; b, coronary section through the heart. The arrows indicate the points and direction of regurgitation.

Coexistence of Organic and Functional Insufficiency.—As has been stated above (page 226), Koester, Krehl, Geipel, and others have shown that the occurrence of vegetations upon the valves is often, perhaps usually, accompanied by foci of myocarditis in the papillary muscles and in the ring of musculature about the mitral orifice. It is the weakening of these muscle-fibres especially which gives rise to the functional insufficiencies, and it is therefore probable that in many cases of organic mitral insufficiency the element of superadded muscular insufficiency is a very important one. Indeed one often meets with persons who, in spite of medium-sized vegetations, suffer little or no discomfort as long as the heart muscle is in good condition, but in whom cardiac symptoms occur as soon as overstrain, anæmia, or febrile disease weakens the myo-

cardium. In the periods of apparent health, the leak is confined to the streams about the edges of the vegetation. In the added functional insufficiency it also takes place at other points along the line of closure. However, it is impossible to differentiate clinically between the organic and the functional elements, and their relative importance in a given case cannot be accurately estimated.

Atheroma of the Mitral Valve.—Atheromatous and calcified patches along the face and edges of the cusps of the valves are also not uncommon (see Fig. 275, page 469). These changes are particularly frequent along the line of closure, where, as shown by Roy and Adami, mechanical injury, hemorrhages, and exudates are most frequent. Pathologically they are brought about by processes similar to those occurring in the walls of the arteries during arteriosclerosis, and the condition is frequently associated with extensive sclerosis of the coronary arteries. Calcified plaques may also be present in the myocardium (case of J. L., Fig. 275). The mechanical effect of such thickenings and areas of rigidity is to prevent the cusps from accommodating themselves to one another, giving rise to leaks which are undistinguishable clinically from those due to vegetations.

Hemorrhage in the Mitral Valve. Occasionally hemorrhages occur in the cusps of the mitral valve, especially after trauma to the chest (Krabs) or labor (Weber and Deguy), and in new-born infants (Fahr). It is probable that the organization of the clot initiates a fibrosis which leads to mitral stenosis.

Functional Mitral Insufficiency.—When the heart muscle attains a certain degree of weakness, leaks at the mitral orifice may take place. They may occur as the direct result of acute cardiac dilatation from a primary cardiac overstrain, though they arise more frequently in hearts whose myocardium has already undergone degenerative or fibrous changes but whose valves are still intact. This functional insufficiency is often seen in cases of myocarditis. On the other hand, functional insufficiency of the mitral valve may arise as a secondary manifestation in organic disease of the aortic valves. This occurs especially when the leak attains a severe grade or the work of the heart is too much increased, the walls of the ventricle and the papillary muscles becoming overstretched during diastole. This phenomenon will be further discussed in the chapter upon aortic insufficiency.

Types of Functional Insufficiency.—The leaks occurring at the mitral valve as the result of muscular weakness may be divided into two groups:

1. Papillary Insufficiency. Leaks occurring at one or more points along the valve (Fig. 180, B, b) when the weakness of a papillary muscle or stretching of a chordæ tendineæ allows the corresponding portion of the valve to be lifted a little, and a small regurgitant stream to emerge at one of the points of puffing and puckering along the line of closure. This may be termed the type of *papillary insufficiency*, and like many cases of uncomplicated organic insufficiency may correspond to the escape of only a small quantity of blood. This papillary insufficiency represents the mildest form of functional regurgitation. The mitral valve is pushed upward during systole, like a sail, impelled by the full force of the entire ventricular wall. This is antagonized by the pull of the relatively small papillary muscles. It is evident that fibre for fibre the papillary muscles incur a much greater strain than the fibres in the ventricular wall, and consequently they are often the first to weaken. When they weaken apposition of the cusps is impaired and regurgitation sets in. Moreover, since T. W. King and Gibson have shown that the normal mitral valve usually holds a less dilatation sets in, it is evident that the papillary type of insufficiency is the only form of functional insufficiency that can occur in a heart that is not greatly dilated.

2. Relative Insufficiency. The second type of functional insufficiency is met with when the *left ventricle is dilated to such an extent that the mitral orifice becomes larger than the available area of valve surface*, and what may be correctly termed a *relative insufficiency* results. Under

these conditions the leak occurs not at a single point but all along the line of closure (Fig. 180, C). The amount of blood which regurgitates under these conditions may be very large.

Pathogenesis of these Types.—The mode of occurrence of these types of insufficiency may be readily demonstrated by the method of Gad and Meigs, though care must be taken that the heart used for the experiment is not in a condition of rigor mortis. If water is forced into the ventricles at various pressures, it will be seen that, as shown by G. A. Gibson, leakage will occur at a relatively slight pressure. This regurgitation will be small in amount, and will be seen to be of the papillary type, but if the pressure is sufficiently increased and the ventricle dilated, the cusps of the valve stand apart, the true relative insufficiency (type 2) is produced, and a large amount of fluid regurgitates. The rôle which stretching of the muscle plays in this regurgitation may be shown by boiling the heart and thereby shortening the fibres in heat rigor, after which the leak that was present disappears and does not recur until much greater pressures are resorted to. Gibson has caused the leak to disappear by tightening a ligature about the mitral muscular ring. It is therefore evident that the tonicity of the cardiac muscle is an important element in determining the occurrence and the degree of leak both in functional and in organic mitral insufficiency. This point is of the greatest importance in therapeutic considerations, and will be referred to later.

Occurrence of Functional Insufficiency.—Functional insufficiency of the mitral valve occurs in primary cardiac overstrain, in anæmias, during the course of and convalescence from infectious diseases, and in many cases of aortic disease. A certain percentage of the cases in which aortic and mitral insufficiencies are found simultaneously belongs to this group.

Lian, in François-Franck's laboratory, has shown that the contraction of the ring of muscle about the mitral valve (mitro-aortic ring) narrows the diameter of the orifice during systole. He demonstrated that when contraction is weakened the valves may not remain in perfect apposition.

Mechanics of the Circulation in Mitral Insufficiency.

It is almost axiomatic to state that the systolic regurgitation of blood from left ventricle to left auricle in mitral insufficiency is accompanied by a fall of pressure in the former and a rise of pressure in the latter.

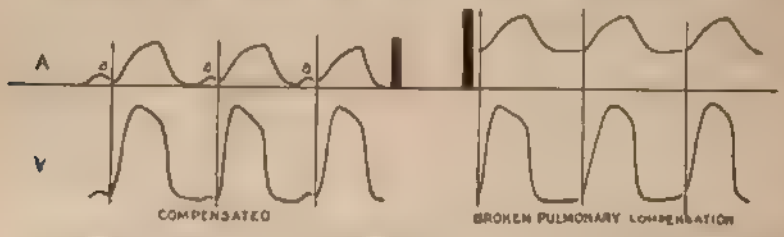

Fig. 181.—Diagram showing the volume and pressure curves under these conditions. (Schematic.) Upper curve, A, intra-auricular pressure, round black line, volume curve of the left auricle; lower curve, V, intraventricular pressure.

The regurgitation of blood into the left auricle causes a systolic rise of pressure to take place in this chamber, replacing the systolic fall of pressure which is present under normal conditions (Fig. 181). The pressure curve within this chamber in mitral insufficiency thus resembles that seen in the right auricle in tricuspid insufficiency—a rise throughout ventricular systole with a fall during diastole, and a small rise when systole of the auricle takes place.

The pressure conditions within the pulmonary circulation are of the greatest importance, and in this both the force-pump and the suction-pump actions of the ventricle show themselves.

Effect of Mitral Insufficiency without Increase in the Strength of the Ventricle.—If the force of the left ventricle remains unaltered after the production of the insufficiency, it stands to reason that less blood will reach the arteries and pass on to the capillaries and systemic veins than did so before. The arterial blood-pressure will fall. Consequently less blood will enter the right side of the heart from the venæ cavæ, and the pressure in the latter will be lowered. The systolic output of the right ventricle will thus be diminished and the pressure in the pulmonary artery will fall. On the other hand, the regurgitated blood in the left auricle and pulmonary veins added to that coming on from the pulmonary artery will cause the pressure in the left auricle, the pulmonary veins, and the capillaries of the lungs to rise (Fig. 183, III).

FIG. 182.—Curve of intraventricular pressure in mitral insufficiency produced on a mechanical model. After Marey. *P V* intraventricular pressure, *P R* arterial pressure. The horizontal line denotes the production of insufficiency. *O* notch due to the auricular systole, *e*, summit of the curve during systole of the ventricle.

Pulmonary Stasis.—As v. Basch and his pupils have shown, congestion of the pulmonary capillaries is the most important cause of cardiac dyspnœa (broken pulmonary compensation). Hence it will not be surprising that dyspnœa from this cause is an early and important symptom of mitral insufficiency, and that its disappearance depends upon other factors which tend to deplete the pulmonary capillaries. (Diminution in the amount of blood entering right ventricle, or weakened suction-pump action of left.) The capillary area is sufficiently elastic to accommodate a considerable amount of regurgitant blood before this furnishes an obstruction to the pulmonary artery, just as is the case with the capillaries of the splanchnic area, but after a time or in severe lesions the intrapulmonary stasis finally makes its effect felt in the pulmonary artery. The pressure there rises (Gerhardt). When the pulmonary stasis becomes extreme the right ventricle, too, becomes overloaded and dilated. Broken systemic compensation sets in (Fig. 183, IV). Stasis occurs in the systemic veins, œdema and ascites take place, and a secondary functional insufficiency of the tricuspid valve may usually be demonstrated. With the occurrence of this secondary leak at the tricuspid orifice, less blood is pumped into the pulmonary circulation, the congestion here diminishes, the dyspnœa dimin-

ishes also, and in spite of the increased gravity of the condition the patient may experience some temporary relief from his symptoms. This phenomenon was noted by T. W. King in 1837, and was designated by him "the safety-valve action of the tricuspid valve." The relief is, however, only transitory, as the accumulation of CO_2 in the blood soon gives rise to dyspnœa from stimulation of the respiratory centre in the medulla, and the real state of cardiac failure manifests itself. If the condition is allowed to continue, the outcome is death.

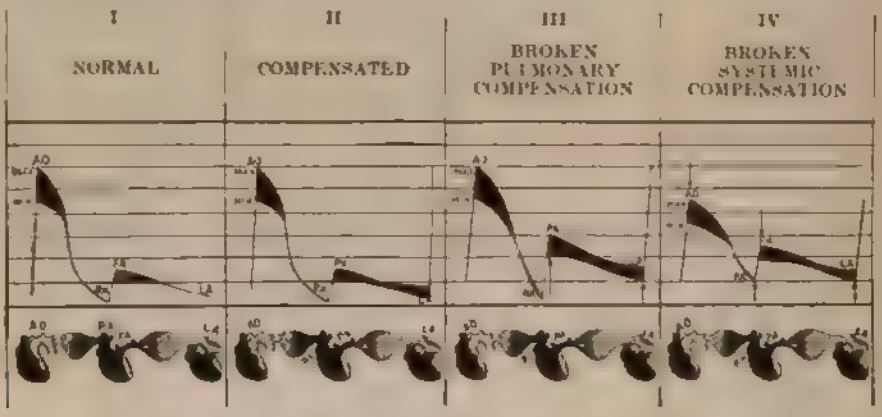

FIG. 183.—Diagram showing the effects of mitral insufficiency upon the circulation. I. Normal. II. Compensation through filling of the auricle in systole but complete emptying in diastole. III. Broken pulmonary compensation. IV. Broken systemic compensation.

Effect of Increase in the Strength of the Left Ventricle.—If, on the other hand, the force of the left ventricle increases, it can throw more blood out into the aorta and arteries; and this blood must first be drawn from the left auricle and pulmonary circulation. So that if the output from the ventricle increase, it will soon be pumping more blood into the aorta than the right ventricle (whose force has remained practically unchanged) pumps into the pulmonary artery. Accordingly more blood leaves the lungs than enters them, and the engorgement passes off. The whole condition may be summed up by the statement that a weakly acting left ventricle overfills the lungs with blood, while a strongly acting left ventricle bails them out. The whole of pulmonary engorgement is, as rightly claimed by v. Basch, a problem not of the right ventricle, but of the left.

CLINICAL MANIFESTATIONS

MITRAL INSUFFICIENCY WITHOUT SYMPTOMS.

The earliest circulatory disturbance in mitral insufficiency is stasis in the pulmonary capillaries, which, as v. Basch and his pupils have shown, leads to cardiac dyspnœa. Accordingly, it is not surprising that dyspnœa should be one of the earliest symptoms of mitral insufficiency. But the

pulmonary stasis does not occur or does not persist when the action of the left ventricle is sufficiently vigorous, and hence in the milder cases shortness of breath may be absent for a long time after the actual formation of the lesion and may make itself evident only upon exertion.

Thus, a young friend of the writer, a boy of eleven, in 1907 contracted tonsillitis accompanied by the typical signs of mitral insufficiency. He was kept moderately quiet on account of the throat lesion and had apparently recovered completely, so that the family did not notice any abnormality until a year afterward, when he became somewhat short of breath upon walking uphill. Since a little caution has been exercised against fast walking he no longer becomes short of breath and remains perfectly free from symptoms. The prognosis is very favorable. In some more vigorous individuals the lesion does not manifest itself at all. The writer has in mind a young man of twenty-one who has shown signs of mitral insufficiency for eight years, but during that period has excelled at football, wrestling, and all the severe forms of sport, also a professor of forty who has had a mitral lesion of rheumatic origin for some time without the slightest physical inconvenience. These cases, though scarcely to be regarded as the rule, are encountered with great frequency in patients who are examined for some other cause. Occasionally such persons have been informed of their trouble, sometimes given a grave prognosis, and come to the physician in great mental distress because they have been told that they have "organic heart trouble," and yet they may reach middle or even old age without serious inconvenience. It is not extremely uncommon to find cases in whom a mitral lesion has been present thirty or forty years without greatly affecting the patient's activity or enjoyment of life.

Pulmonary Complications of Mitral Disease. The prolonged stasis and high pressure in the pulmonary capillaries may, however, give rise to permanent changes in their walls and in the tissues about them and facilitate the occurrence of a chronic bronchitis. This bronchitis resulting from stasis may be considered analogous in origin to the ulcerations and weeping eczema found upon the legs in association with varicose veins or cardiac œdema. The presence of this chronic bronchitis is liable to arouse a *suspicion of tuberculosis* when the trouble is really cardiac. The suspicion is sometimes still further aroused in the cases in which the walls of the capillaries in some area of the lungs have become eroded and occasional pulmonary hemorrhages take place. Thus, Osler and A. G. Gibson mention the case of a physician who suffered from occasional *haemoptysis* due to mitral insufficiency over a period of twenty-five years, each attack giving rise to great relief of his cardiac symptoms by relieving the engorgement of the left auricle. The diagnosis in such cases is often difficult, but may be made when there is continued absence of tubercle bacilli from the sputum, and especially from the ejected blood, associated with the signs of a definite mitral insufficiency. Further confirmation may be gained by a negative cutaneous or ophthalmic reaction with tuberculin, or if necessary by a negative subcutaneous injection of the latter.

MITRAL INSUFFICIENCY WITH SYMPTOMS.

Second Stage of Mitral Insufficiency (Broken Pulmonary Compensation).—A more severe stage with less favorable prognosis is that in which the lesion manifests itself by subjective symptoms. As in most other cardiac disturbances, palpitation occurs early in the disease. Palpitation is, however, common in healthy persons, and, as shown by Hirschfelder (see page 157), cannot as yet be regarded as signifying functional weakness, so that its presence scarcely suffices to direct suspicion to the cardiac valves.

The earliest symptom of real importance is *shortness of breath*. This occurs at an earlier stage in affections of the mitral than of the aortic valves, but is more marked in early mitral stenosis than in early mitral insufficiency. Nevertheless, it may be *quite severe in comparatively mild grades* of the latter, especially on exertion. As has been stated above, the dyspnœa is due to the pulmonary engorgement, as

shown by v. Basch. It is therefore liable to pass off when the left ventricle hypertrophies and the systolic output is increased and the suction pump action is increased. For practical purposes, therefore, such persons whose cardiac function has been restored by the hypertrophy of the left ventricle may be said to have passed from the second stage insufficiency back into the first. They often remain in this excellent condition for a number of years, and sometimes oscillate between the first and the second stages for a number of years more.

Tonicity of the Heart as a Factor Governing the Leak.—A most important factor in preserving this balance is the tonicity of the cardiac muscle, for when the tonus is maintained the cusps of the valve are approximated as tightly as possible about the thickening, and the amount of leak is reduced to minimal, whereas when the tonicity is low the organic leak is further supplemented by a papillary or a relative insufficiency. A small leak is thus, if only for a time, transformed into a large one. When tonicity is low it is harder for the heart to recover from such an additional strain than if the latter occur at a time when the tonus is increased. The added functional insufficiency may thus become permanent. We have here another example of the vicious circle:

Leak at mitral valve
Leak increased
↑
Papillary or relative insufficiency ← Heart strain
Diminished tonicity

It is evident, therefore, that in spite of the comparative ease with which patients may recover from the symptoms of mitral insufficiency when the case is placed under favorable conditions as soon as possible, yet the case may rapidly become a grave one if these precautions are neglected, so that, in the words of Osler, mitral insufficiency may be either the mildest or the gravest of valvular lesions.

Third Stage of Mitral Insufficiency (Broken Systemic Compensation).—The second stage of mitral insufficiency represents the physiological conditions shown in Fig. 183, IV, when the stasis is in the lungs and the work of the right ventricle is gradually increasing. The third stage represents that in which the right ventricle also has begun to fail, and blood begins to stagnate in the systemic veins as well. The pressure in these veins increases two- to threefold (from 5–8 cm. H_2O to 20–30 cm.) as can be shown by the method of Eyster and Hooker. As a result they dilate and the flow through them is slowed, changes occur in the capillary walls, and œdema soon takes place—first in the feet, later in the shins, thighs, genitalia, and back. With the onset of these manifestations the shortness of breath becomes extreme, a dyspnœa of medullary origin adding its effect to the pulmonary engorgement. The patient is compelled to sit up all the time, gasping for breath, occasionally with paroxysms of real cardiac asthma and palpitation, sometimes with pains in the heart, severe cough, and expectoration of considerable amounts of sputum which often contains cells loaded with blood pigment (Herzfehlerzellen). The urine becomes scant and loaded with albumin and casts. Ascites may set in and may

even become so great as to require repeated tapping. The same is true of hydrothorax. On this account it is more common upon the right side. Unless the course of the disease is checked, death may follow after this stage has set in, but if the work of the heart can be diminished and its action strengthened it may soon pump out the stagnating blood and lower the venous pressure. From this cause, and owing to the concomitant increase in cardiac tonicity, the dilatation diminishes. The element of regurgitation which is of functional origin disappears and the amount of blood regurgitating is once more reduced to that which flows past the vegetations. The work of the heart is again brought to its minimum, and thus it is that *almost unhoped-for recoveries* may occur in mitral insufficiency when properly treated.

PHYSICAL EXAMINATION.

Inspection.—The typical picture of mitral disease is seen in the flushed pink cheeks with slight tinge of purple, slightly dilated venules, and bright watery eyes, giving on superficial inspection the appearance of superabundant health—the so-called *mitral facies*. This is in sharp contrast to the pale, pasty, or sallow color of aortic insufficiency, or the livid purple of emphysema. The lips show a moderate cyanosis. There is, as a rule, no special throbbing seen in the carotids. The venous pulse is usually well seen and is of the normal "double" type. Since the disturbance of function in the first two stages is in the pulmonary circulation and not in the systemic, no change in the jugular pulsation is to be found nor would be expected until the third stage, when the onset of tricuspid insufficiency causes it to assume the "single" or ventricular form.

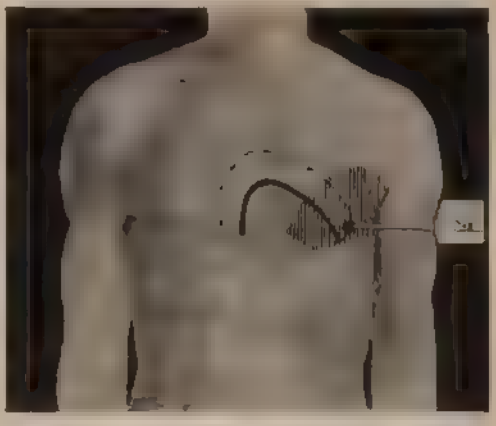

FIG. 184.—Distribution of the murmur in mitral insufficiency. Distribution upon the chest wall. Light line outline of cardiac dulness; heavy line outline of cardiac flatness. Shaded area shows distribution of the murmur. The diagram at the right indicates the relation of the murmur to the cardiac cycle.

The chest may show *precordial bulging*, especially in children and in cases of long standing. The apex impulse if visible is *displaced outwards toward the axilla rather than downwards*. It is frequently of a slow heaving character, while in cases of long-standing pulmonary stasis there may be also a wavy systolic retraction of the interspaces which mark the hypertrophy of the right ventricle. Occasionally a systolic impulse in the second left interspace shows the vigorous pulsation of the pulmonary artery. In very large hearts it is not uncommon to find a systolic retraction present along the outer border of dulness, even when adherent pericardium is absent.

In long-standing cases changes in the extremities also take place, especially slight clubbing of the fingers.

Palpation usually reveals a strong, slow, heaving impulse with a more or less intense thrill lasting throughout the period of systole. This thrill is probably due to the impact of eddy currents passing by the vegetation and striking the heart wall, whose impact sets the valve into vibrations that are communicated along the chordæ tendineæ to the walls of the heart and then to the chest. It is most intense at the apex, but is often palpable over the entire precordium, occasionally in the axilla, and sometimes at the back in the left interscapular region. The thrill at the back is rarely felt except in children and thin-chested persons, but as it represents the direct impact of regurgitation it is very characteristic. In a few cases the thrill from a mitral lesion can be felt in the vessels of the neck, and sometimes also over the entire chest.

Fig. 185.—Cross section of the body showing how the thrill and murmur reach the chest wall. The heavily stippled areas indicate the areas over which the murmur is heard.

The shock accompanying the second heart sound is practically unchanged, though it is often more distinct, especially over the pulmonary area.

Percussion. The characteristic cardiac outline in mitral insufficiency shows an increase in area horizontally (M L) towards the left, which is sometimes enormous and may reach well into the axilla and as much as 22 cm. from the midline by percussion. When the apex approaches the axillary convexity, as shown by Moritz, the area of dulness usually extends somewhat beyond the actual outline of the heart; but with the lightest possible percussion (the threshold percussion) this error may often be reduced to a centimetre or less. In contrast to the cardiac outline in aortic insufficiency, the left border of dulness or of the shadow seen with the X-ray is increased diagonally upwards and outwards, as well as outwards in the horizontal diameter (Fig. 186). The upward extension may reach the second rib and pass outward into the second left interspace. According to the autopsy findings of Harris, this is not due to the dilatation of the left auricle, but to the increase in size of the conus arteriosus of the right ventricle and to some dilatation of the pulmonary artery.

Fig. 186.—Radiograph of a patient with mitral insufficiency, showing horizontal enlargement of the heart to the left. After Bougeault and Schittenhelm.

Harris has shown that even an extremely dilated left auricle is not visible from the front of the chest under these circumstances. As is therefore to be expected, such dilatations correspond to the later rather than to the earlier stages of the disease, and are also

more common in mitral stenosis than insufficiency. The cardiac dulness is not increased to the right until the third stage of the disease.

The Systolic Murmur. The data obtained from auscultation usually furnish the basis for the diagnosis of mitral insufficiency. The characteristic sign is the presence of a murmur heard at the apex and in the left axilla throughout systole, *i.e.*, lasting up to the second sound. The sounds of different systolic murmurs are described by the French writers as resembling the rasping of a file, the sawing of wood, the hissing of a jet of steam, the cooing of a dove! These variations depend upon many sound-producing factors so complex that it is impossible to predict the characteristics of the lesion upon the valve by the murmur to which it gives rise. In general, rough, roaring, sawing, and purring murmurs are very often produced by thickened or calcified vegetations, which act more or less as sounding-boards, while whispering or blowing murmurs are produced by regurgitant streams passing over smooth-walled valves, and occur especially in functional insufficiencies. However, exceptions to this are frequent.

FIG. 187.—Diagram of Fig. 186, showing the directions in which cardiac enlargement has taken place. The broken line represents the outline of the normal heart. The arrows indicate the contingates (M. and I.) which are most enlarged.

Digital Imitation of the Mitral Systolic Murmur.—The mitral systolic murmur can be reproduced by Lamed's or Smith's methods by a stroke across the elbow or across the back of the hand. To imitate a murmur accompanying the first sound the latter should be produced by a slow flexion of the finger which combines the blow and the stroking. For demonstrations to a group of students these manœuvres may be carried out upon a derby hat. The stroke should be of long duration and should be followed immediately by the tap which represents the second sound.

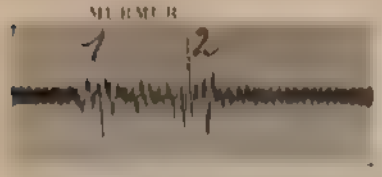

FIG. 188.—Graphic records of the heart sounds, showing the systolic murmur. (Kindness of Prof. Einthoven.) 1, 2 heart sounds. The vibrations between 1 and 2 and some of those composing 1 are due to the murmur.

Excellent phonographic tracings of this murmur have been made by Einthoven as well as by Weiss and Joachim. These observers have shown that in contrast to the murmur of aortic stenosis the murmur of mitral insufficiency always begins synchronously with the first sound. It is loudest at the time of the first sound, which it may entirely replace and may then be uniform throughout the period of systole or take on a decrescendo character, in contrast to the aortic murmur which follows the first sound and is crescendo in character.

They have shown further that in some cases the first sound and murmur precede the carotid wave by a greater interval than normally (lengthening of the presphygmic period from .08–.10 sec. to .12–.15 sec.). At the very beginning of systole blood rushes back into the auricle. This escape causes the pressure in the ventricle to rise more slowly than usual and hence aortic valves open a little later. Hence the presphygmic period is

later. It is possible that the duration of the presphygmic period may serve as a guide to indicate the degree of leakage, shortening indicating an improvement, lengthening an increase in leakage. However, other observers find lengthened presphygmic periods from different causes.

In the accidental murmurs of anæmia Weiss and Joachim have found a normal presphygmic period.

It is stated by some writers that functional murmurs occur late in systole, mesosystolic or telesystolic, because comparatively little regurgitation occurs until the intraventricular pressure has reached its maximum and the papillary muscles begin to weaken. There are no graphic records supporting this claim, and the subject demands experimental investigation rather than clinical speculation.

As regards distribution, the murmur of mitral insufficiency is heard over those areas of chest wall which are nearest to the left ventricle and left auricle, namely, the apex and the neighboring areas to the left (Fig. 184).

The reason why the murmur of mitral insufficiency is heard so loudly at the apex is not perfectly clear, since this propagation is opposite to the direction of the regurgitant stream. It is possible, however, that the vibrations of the mitral valve may be transmitted to the anterior papillary muscle and apex by the papillary muscles which act like violin strings. This would also explain why the murmur is so loud in the anterior part of the left axilla at a considerable distance from the left auricle but near the anterior papillary muscle (Fig. 185).

In children and in many persons with small, thin-walled chests the murmur is also heard over the left interscapular region to which it is transmitted directly from the left auricle. This distribution is the one which is most typical, since it represents transmission in the direction of leakage, and the comparative infrequency with which it is heard is due entirely to the thickness of the chest wall and the layer of lung. Certain excellent German observers, however, are unwilling to diagnose mitral insufficiency unless this murmur is present.

In order to obviate these disturbing factors, August Hoffmann and later Gerhardt have attempted to use the oesophageal auscultation described on page 104, but it is unpleasant to the patient, and, besides, the murmur may be no more distinct than at the apex.

Another rarer site for the systolic murmur of mitral insufficiency, as shown by Naunyn, is the pulmonary area—the second and third left interspaces at the sternal margin and as far out as the parasternal line. Naunyn thinks that in this region the vibrations are communicated by the hypertrophied and dilated left auricle to the pulmonary artery. This murmur must always be carefully differentiated from the accidental pulmonary murmur heard in this region and from that due to pulmonary stenosis and sclerosis.

Over the right ventricle, that is from the left parasternal line to the left sternal margin, the murmur of mitral insufficiency is usually heard, diminishing in intensity as the distance from the apex increases.

Differentiation from Accidental and Tricuspid Murmurs.—This is in sharp contrast to the accidental or "haemic" murmurs which are loudest over the right ventricle and especially over its upper portion (in the second and third left interspaces). These murmurs diminish over the apex while the mitral murmur increases. They are rarely heard to the left of the apex. They are later, softer, shorter, more superficial, they vary with inspiration, expiration, and change of position; and the heart is, as a rule, not hypertrophied. On the other hand, there is no reason to believe that the distribution of the murmur due to functional mitral insufficiency differs from that due to vegetations upon the valves, and it is frequently impos-

sible to differentiate between the two conditions.[1] The common statement that murmurs of organic origin are transmitted to the axilla while those of functional origin are not is due to a confusion of "functional" with "accidental" murmurs. All loud and rough murmurs are transmitted further than soft ones, and murmurs due to vegetations are usually louder than functional murmurs, but a soft murmur of organic origin is probably no louder than many murmurs of functional origin.

The murmur due to tricuspid insufficiency is heard loudest over the lower portion of the sternum, the epigastrium, and often also to the right of the sternum. That of aortic sclerosis or aortic stenosis is loudest over the second and first right interspace, and is usually transmitted to the carotid and axillary arteries. The same applies to the murmur due to aneurism. Moreover, as stated by Boy-Teissier, it is always meso- or telesystolic, and therefore follows but never replaces the first sound.

The Second Sound.—The second sound at the apex and over the aortic area shows no special change, but over the pulmonic area it may be greatly accentuated. This is not always the case, because, as seen in Fig. 183, the pressure in the pulmonary artery is not always increased; but during the course of a mitral insufficiency the intensity of the second pulmonic may change. In interpreting the significance of this change it must be remembered that rise of pulmonary pressure may occur either when the force of both ventricles is increasing or when extreme engorgement of the pulmonary vessels has occurred. In the former case it will be accompanied by the general signs of improvement in the left ventricle, larger pulse, increased maximal blood-pressure, increased pulse-pressure; in the latter case by failure of the left ventricle. Regarding the rôle which vasomotor changes in the pulmonary vessels play under clinical conditions little is known; but they also can affect the intensity of the second pulmonic.

Pulse.—The pulse in mitral insufficiency shows no characteristic changes. Dependent upon blood-pressure, systolic output, and pulse-pressure which vary considerably, it may be large or small, hard or soft. Dependent upon the condition of the heart muscle and especially of the left auricle, it may be regular or irregular. The condition of the individual patient at any single stage of the disease must be viewed in the light of these determining factors.

Blood-pressure.—As has been seen above, the sudden production of mitral insufficiency both in animals and on the model is followed directly by a fall of blood-pressure and diminution of pulse-pressure, unless the strength of the ventricle and its systolic output be increased. Such an increase does take place, however, during hypertrophy, and hypertrophy of the left ventricle is the rule in mitral insufficiency. Moreover, when the output into the arteries is diminished by the backflow into the left auricle, these vessels undergo a compensatory constriction and narrow the blood channel. However, as the left ventricle hypertrophies, its systolic output increases at the expense of the residual blood. These two compensatory factors combine to maintain the blood-pressure and pulse-pressure at their original

[1] The confusion of the terms "hæmic" (accidental) and "functional" murmurs so common in clinical notes seems therefore to be both unnecessary and misleading, and should be carefully avoided.

level, and at times even exceed it. For example, a prominent medical educator who has a mild leak at the mitral valve but suffers no symptoms, has a maximal pressure of 140, a minimal of 95, and a large full pulse. The compensation in this case has more than balanced the disturbance in the circulation. This increase of pulse- and blood-pressure is not always indicative of improvement, but may occur also as terminal events under the stimulation of medullary asphyxia (see page 27). On the other hand, the presence of a rather small pulse and rather low blood-pressure and pulse-pressure may merely represent the natural effect of the lesion unaltered by compensatory changes on the one hand, or on the other may represent the failure of the left ventricle to maintain the circulation. The presence of arteriosclerosis may in itself tend to modify the blood-pressure, and to increase a blood-pressure and pulse-pressure that would otherwise be small. In any case the blood-pressure shows no characteristic features in mitral insufficiency, and the figures obtained are to be viewed as the algebraic sum of various circulatory factors, rather than as absolute measures of cardiac vigor. It must be remembered further that the cardiac symptoms are due mainly to changes in the pulmonary circulation, while the blood-pressure changes are concerned only with the systemic.

Arrhythmia.—The action of the heart is often irregular in mitral disease, so that the arrhythmia in these conditions is frequently described as "the mitralized pulse." Physiologically, the "mitralized pulse" represents an irregularity probably due to numerous extrasystoles arising in the left auricle but so frequent as to disturb the rhythm completely. In contrast to the absolute irregularity arising in the right auricle, the *a* wave upon the jugular tracing may persist in spite of the arrhythmia, for the right auricle may not be paralyzed. Joachim has shown by œsophageal tracings that the left auricle is often paralyzed under such circumstances. With the irregularity there are often very feeble early extrasystoles too weak to open the aortic valves and to cause a pulse wave. The second heart sound is lacking with these beats because the valves are not opened.

The origin of the irregularity is probably in the distended left auricle (cf. also page 75). It is sometimes, but by no means always, accompanied by paralysis of the auricle, as shown by the venous tracings, but the exact mechanism by which this form of irregularity is produced requires further investigation in order that its diagnostic and prognostic significance may be thoroughly understood.

The condition of the radial, temporal, and other arteries may vary considerably, but, especially in patients above 40, may show considerable grades of arteriosclerosis. This is more common and somewhat more extensive in patients suffering from any cardiac disease than in persons with normal hearts (Wild).

Lungs.—Examination of the thorax and lungs in mitral insufficiency reveals the usual signs corresponding to the pulmonary changes described above—small, moist, and piping râles corresponding to the bronchitis often associated with hyper-resonance on percussion. Over areas of hydrothorax there are absolute flatness on percussion, absence of vocal fremitus and breath sounds, Koranyi's flatness over the lower thoracic spines, and Grocco's triangle, paravertebral dulness to the left of the midline.

Abdomen.—The abdomen rarely shows any special change during the milder stages of the disease. Occasionally one finds, as in a young girl recently under the writer's observation in the Johns Hopkins Dispensary, the remains of an old splenic infarction, characterized by enlargement, hardness, and tenderness of the organ. This may persist for some months. Enlargement and pulsation of the liver and ascites (portal stasis) belong to the stage of broken systemic compensation (see page 338). The same also applies to œdema of the feet, ankles, and legs, which occurs in the mildest form of broken compensation. These phenomena, though of serious import, are by no means harbingers of death, for with proper treatment many cases outlive one or even several breaks in compensation for many years.

The following represent the course of typical cases of mitral insufficiency.

CASE OF MITRAL INSUFFICIENCY.

C. H., ship carpenter, aged 63, first admitted to the Johns Hopkins Hospital in November, 1899, complaining of shortness of breath. Family history negative. The patient has always been a robust man. He gives a doubtful history of rheumatism, but a definite history of pneumonia five years before admission. No venereal history. He has always been a hard eater, hard drinker, and a hard worker.

The present illness began with attacks of paroxysmal dyspnœa upon exertion eight or nine months before admission. Two months before admission an attack came on spontaneously while in a warm room. During the past month he has not been able to lie down in bed owing to dyspnœa and the onset of a smothering feeling. He has had no cough, no hæmoptysis. Examination on this admission showed a stout, well-nourished man with mucous membranes a trifle purple and dilated venules over the face. His chest was barrel shaped and there was a little fluid (flatness and impaired breath sounds) at the bases behind.

Heart.—Apex was situated in the 6th interspace 16.5 cm. from the midline. The area of cardiac dulness reached upward to the second costal cartilage and 5 cm. to the right of the sternum. There is a well-marked systolic murmur heard over the body of the heart and over the anterior part of the axilla. The second sound is everywhere clear; the second aortic booming.

The liver is slightly enlarged, being just palpable; the spleen is not. The abdomen is full, the flanks bulge, and there is slight movable dulness in the flanks. Genitalia normal. There is slight oedema of the ankles. Blood count normal. Urine is dark sherry colored, specific gravity 1024, acid, contains a small amount of albumin and some hyaline and granular casts.

The patient was put to bed on soft diet, given daily purgation with magnesium sulphate (30 Gm., ℥i), also 8 doses of tincture of digitalis (1 c.c., ♏xv) at intervals of four hours, followed up by strychnine 1.5 mg. He was also given potassium iodide 1 Gm. (gr. xv) after meals.

Œdema disappeared and orthopnœa also, so that within ten days the patient could sleep with his head low and could walk without dyspnœa. He then left the hospital.

He was next seen three years later, having been perfectly well until he took cold one month before, since when he had shortness of breath on exertion and on lying down. He had some cough and slight swelling of the feet. The physical condition was about as on the first admission, except that the systolic murmur entirely replaced the first sound and was well heard in the axilla. Maximal blood-pressure 182 mm. Hg. He again improved rapidly and left the hospital in two weeks.

He entered the hospital again one year later with the same signs, the liver being now 2 cm. below the costal margin. Once more he improved rapidly under treatment; the liver receded, and he was discharged, only to be readmitted in the same condition five weeks later, when symptoms dated from exposure to the wet. He then had some tenacious sputum streaked with blood and numerous moist râles were heard everywhere over his chest. Recovery was once more uneventful.

COMPLICATIONS AND SEQUELÆ.

There are few complications and sequelæ which are more characteristic of mitral insufficiency than of other valvular diseases. Those symptoms due to pulmonary engorgement bronchitis, hæmoptysis, and pulmonary œdema have already been discussed. Embolism from loosening of vegetations upon the mitral valve or of clots which have formed in the left auricle during periods of stasis is an occasional occurrence, especially in severe cases, but less common than in mitral stenosis. As the result of this there may be the production of infarcts in the various organs spleen, kidneys, and brain and of ecchymoses in the skin.

In contrast to mitral stenosis, pulmonary tuberculosis occurs in mitral insufficiency with the same frequency as in otherwise normal individuals (Meusenburg).

As may be seen in Fig. 166, mitral insufficiency is very frequently associated with other valvular diseases, 29 per cent. of all the cases of valvular disease at the Johns Hopkins Hospital being accompanied by aortic insufficiency, 21 per cent. by mitral stenosis. In these cases the mitral insufficiency is sometimes the original lesion, the other lesion resulting from a metastatic infection or subsequent organization. On the other hand, the mitral insufficiency associated with aortic insufficiency may also be a functional one due to overfilling of the ventricles. The mitral insufficiency which appears late in the course of mitral stenosis is due to the inability of the thickened valves to close. Coronary sclerosis, as shown by Wild, is more than usually common in chronic valvular disease, and hence should be borne in mind in establishing the prognosis.

Pericarditis is one of the common complications, especially in children, in whom adhesive pericarditis is to be feared.

Since the majority of cases of mitral disease are of rheumatic origin, diseases of the rheumatic cycle, tonsillitis, articular rheumatism, chorea in children, and affections of the urticarial group, are particularly common. Of these arthritis is the most frequent as well as the most stubborn and dangerous.

TREATMENT.

The management of cases of mitral disease does not depart in any essential particular from the general type of treatment of cardiac disease. It should be directed to three ends:

1. Removing the overstrain;
2. Increasing the strength of the heart,
3. Avoidance of infection, and removal of the foci

In the mildest cases, the insufficiency shown by signs but not by symptoms, due especially to the formation of a new vegetation, it is most important that the cardiac tonicity should be maintained, that the amount of leakage should thus be kept down to its minimum, and that cardiac hypertrophy should be induced before symptoms have set in. It is therefore most important to spare the heart every effort. If the patient is seen at the onset of the disease he should be kept at absolute rest in bed for at least a couple of weeks after temperature has returned to normal and all

signs of acute disease have passed. Too much care cannot be exercised at this time, since this is the crucial epoch in determining the severity of the case. It is important not only to maintain the tonicity of the heart muscle but to preserve the valves from all further injury until the vegetations have become thoroughly organized and lined with endothelium, and the germs have disappeared from the original focus of infection. When the tonsil is the source of infection, it should be completely dissected out as soon as acute infection has passed off, in order to prevent reinfection of the valves from this source. The results obtained in the Medical Clinic of the Johns Hopkins Hospital, where this practice has been carried out at Prof. Barker's suggestion, have been very gratifying. In many cases the recovery from the first attack has been more rapid than had been usual before this treatment had been resorted to (see page 317), and it seems probable that reinfection of the valve is of less frequent occurrence thereafter. Similar results are seen in gonorrhœal endocarditis after treatment of the urethritis.

In these mild cases drug treatment may not be absolutely necessary. In how far Cloetta's suggestion as to the early use of digitalis should be carried out is still unsettled, but at least the administration of strychnine in doses of from 1 to 3 mg. ($\frac{1}{30}$ to $\frac{1}{20}$ gr.) is advisable in order to increase the tonicity of the heart muscle. However, the blood-pressure and hence the strain upon the valves should not be materially increased (not more than 10 mm. Hg), and the dose of strychnine should be reduced if it rise above this level. Digitalis is not necessary in cases of this type.

The bronchitis which frequently accompanies cases of this type does not differ greatly from the ordinary forms of chronic bronchitis, and is associated with the usual pulmonary bacteria found in these conditions—the streptococcus, pneumococcus, influenza bacillus, Friedländer's bacillus, etc. Treatment is therefore the same as for ordinary bronchitis, a soothing steam inhalation being very useful. The following are to be recommended:

Oleum pini sylvestris, or Creosoti, ℥iss;	5
Tr. benzoin. co., Tr. opii camphorat., ää ℥iiss	75

A teaspoonful inhaled with steam from an atomizer, or from a funnel above a glass jar into which a teaspoonful of the remedy and a pint of boiling water have been placed. Codein .015 to .030 Gm. (gr. ¼ to ½) or heroin 2.5 mg. (gr. $\frac{1}{30}$ to $\frac{1}{12}$) may be given by mouth to relieve the cough by reducing bronchial secretion and irritability.

Physical Re-education.—When the stage of acute symptoms has passed the stage of re-education begins. The heart though injured must be trained to perform the day's work without strain. To do this the heart muscle must be stronger than normal; it must have hypertrophied. The process of hypertrophy after valvular lesion, like the heart hypertrophy of an athlete in training, requires time. However, the amount of hypertrophy setting in after a valvular lesion is greater than that after a prolonged period of muscular exercise, and hence may be expected to take a longer time. At this stage Nauheim baths and resisted movements may be dispensed with

as long as a reasonable supervision is kept over the patient. He should never be allowed to become either very tired or short of breath, and six months or a year should elapse before he is allowed to run, participate in games, severe exercise, or manual labor. The current statement that the prognosis is doubtful during the first year after the occurrence of a valvular lesion is due largely to the intercurrence of acute overstrain of the heart muscle before hypertrophy is complete.

The condition is quite different, however, when the mitral insufficiency is of long standing before it is encountered by the physician, as is frequently the case in routine examinations for life insurance, civil service, etc. The disease may then be said to have cured itself already, and beyond gently admonishing the patient against over-exertion no further precautions are necessary. It is often unwise to inform a man or woman of nervous temperament that a heart lesion is present, since worry may in itself contribute to the cardiac overstrain.

Cases of functional mitral insufficiency are rarely devoid of cardiac symptoms, and hence will be considered under the second group.

Treatment of the Second Stage. The second stage of mitral insufficiency, in which dyspnœa and other symptoms of cardiac origin are present, represents a condition of chronic cardiac overstrain. Indeed it is the abnormal severity of these symptoms following some slight exertion which usually calls attention to the existence of the lesion. The treatment does not depart in any essential particular from that which has already been discussed in the case of chronic overstrain of the myocardium. Just as in the milder cases the most important element in the treatment is absolute rest in bed, continued until long after symptoms have subsided. The diet should at first be very light (see page 167), and should be very gradually increased after symptoms have subsided. As in the milder cases, vigorous doses of strychnine (2 to 3 mg. [gr. $\frac{1}{30}$ to $\frac{1}{20}$] every four hours) should be begun at once. The bowels should be kept moving freely by means of Seidlitz powders, Epsom salts, Hunyadi water, Mistura ferri aperiens, or some other mild laxative. It is important that the patient should sleep well at night, and trional (1 Gm. = gr. xv) or some other soporific may be given, if necessary with the addition of codein (15 mg. = gr. ss). If symptoms have not diminished after a couple of days of this treatment, digitalis should be resorted to (see page 178), for it is important not only to reduce the strain but also to strengthen the heart muscle as rapidly as possible in order to prevent the overstrain from becoming permanent. Indeed it may be said that this is the all-important stage in the course of mitral insufficiency and of all other valvular lesions, the stage which determines whether the patient may hope to return to a life of activity or must look forward to one of permanent invalidism, and this question is often decided by the promptness or tardiness with which the symptoms disappear when the patient is at rest. In this stage the primary source of infection or reinfection should be treated just as in the milder ones, but the period of rest should be longer and the period of physical re-education and gymnastics should be very carefully undertaken. The more systematic methods, such as those of Schott, Herz and Oertel, are especially valuable, as are also the Nauheim baths. The important factor,

however, is that, whatever the method of treatment, the patient should never be allowed to become fatigued or short of breath, he should be gradually trained up to his optimum strength, and he should never be allowed to attempt to exceed his limit.

Treatment of the Third Stage.—In the third stage of mitral insufficiency, that of broken compensation, the burden of the cardiac failure has been shifted from the left ventricle to the right. The treatment therefore follows the rules laid down for broken compensation due to any cause whatever: absolute rest, immediate use of digitalis (especially along with nitroglycerin, sodium nitrite, or erythrol tetranitrate), free purgation, and very light diet being the essential features. Hypodermic injections of morphine (8 to 15 mg., gr. ⅛ to ¼) may be necessary, but they should be used with caution, since they decrease the irritability of the respiratory centre and thus lead to accumulation of CO_2 and cardiac asthma. Since many of the symptoms are due to a high pressure in the vena cava and consequent dilatation of the right auricle and ventricle, venesection is often followed by great improvement, and should be regarded as an important therapeutic measure during the acute stage of the overstrain. But in the presence of anæmia it should not be resorted to. Intravenous strophanthin (0.5 to 1 mg.) is of the greatest value when the symptoms have become alarming, and should be followed by the usual course of digitalis, or by daily intramuscular injections of strophanthin.

As symptoms subside, the condition and its treatment pass into those of the second stage, and a gradual return to normal may occur. On the other hand the symptoms may increase, hydrothorax and ascites may become extensive and may require tapping, and the œdema may become extreme. To diminish these it may be advisable to use diuretics, such as theobromin acetate (agurin), theocin, or acettheocin sodium, or else potassium acetate and citrate.

PROGNOSIS.

Exactly how much benefit can be effected by treatment varies with each individual case, and depends upon factors which are difficult to foretell. It is especially true of mitral insufficiency that while there is life there is hope, for the patient may almost completely recover from one or more attacks of broken compensation and yet remain comparatively free from symptoms for a number of years.

As regards the prognosis for the individual attack no absolute rule can be laid down, but much importance may be attached to the rapidity of change for the better or for the worse. A rapid improvement during the first two days may be construed as favorable for the ultimate outcome; a slow recovery usually indicates a severe residuum of trouble; an increase of symptoms in spite of treatment is of grave significance.

Between attacks the patient's ability to hold his own or even to improve his condition depends entirely upon his ability to keep himself free from overstrain and reinfection. In all cases the physician should be somewhat guarded in his statements regarding the future.

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III.

MITRAL STENOSIS.

HISTORICAL.

After mitral insufficiency the most common affection of the mitral valve is that which leads to mitral stenosis. This condition was first described by John Mayow in 1669 in the case of a young man who died after several years of dyspnœa, palpitation, and attacks of syncope. "At autopsy he found dilatation and hypertrophy of the right ventricle and almost complete closure of the left auriculoventricular orifice by a 'cartilaginous product,' so that the blood could scarcely traverse the left ventricle and was compelled to flow backward to the pulmonary vessels, hence the distention of the right ventricle." Vieussens (1715) described similar findings, noting also that the papillary muscles were changed to small short tendons, and that the right ventricle was so dilated as to allow reflux of blood past the tricuspid valve. He further noted weakness and irregularity of the pulse, and ascribed it to "too small quantity of blood which the left ventricle furnished to the aorta . . . and the irregularity of its contractions." Senac (1749) and others described cases, so that the lesion became quite familiar to pathologists in the early part of the nineteenth century.

PATHOLOGICAL ANATOMY AND PATHOGENESIS.

Pathologically, it is brought about by a chronic inflammatory process taking place in the tissue of the valve which leads to thickening and fusion of the cusps. By this fusion the valve comes to form a sort of funnel, sometimes elongated, sometimes short and shallow. The walls are much thicker than those of the normal valve, owing to a chronic infiltration and proliferation of the connective tissue within them. They are usually more or less rigid, sometimes smooth, sometimes rough from remains of old vegetations and calcifications, and the auricular surface is often puckered or thrown into folds. The lower margin of the valve is attached to the chordæ tendineæ, which become shortened and thickened very early in the process. The orifice thus formed is sometimes circular, sometimes oval or button-holed, sometimes irregular (Fig. 189). According to Sansom, the funnel shape is more common in

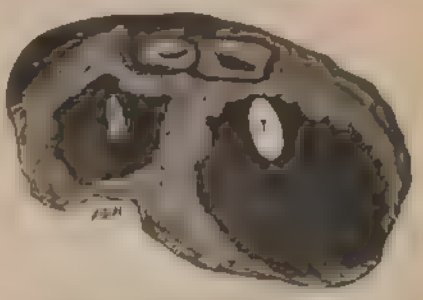

Fig. 189.—Human heart, showing mitral (*M*) and tricuspid (*T*) stenosis. Viewed from above. The auricles have been cut through.

children, while the buttonhole is more common in adults and represents a lesion of longer duration. The orifice may often become very small before death ensues, sometimes barely large enough to admit a goose-quill (Sansom). In some long-standing lesions the margin of the valve may become so thick and stiff (sometimes calcified, cartilaginous, or ossified) that it does not close during ventricular systole; in others the edges become retracted so that they no longer cover the orifice. In either case secondary mitral insufficiency may result.

Occurrence. According to the studies of Lockhart Gillespie, mitral stenosis is most common in women, increasing in frequency up to the age of 20. In men it is scarcely more than half as common, but reaches its maximum frequency a decade later. The males affected die earlier than the females, however; the average period of death being from 30 to 39 with the former, while with the latter it is 40 to 49. The presence of the double mitral lesion does not shorten the average period of life.

Etiology.—As regards etiology, Cheadle, Samways, Duroziez, and Dyce Duckworth agree that rheumatism is the etiological factor in at least 60 per cent. to 78 per cent. of the cases, other acute infections, chlorosis (Goodhart), chronic nephritis, gout, arteriosclerosis, and puerperal infections representing the other etiological factors. Potain also believes that pulmonary tuberculosis is an important factor, but this is disputed by many writers; most of whom agree with Traube that tuberculosis is far less frequent (5 per cent.) in the presence of mitral stenosis than in normal individuals (12 per cent.) or in those with other heart lesions (Meisenburg, Tüeston).

Pathogenesis. — As regards pathogenesis, Huchard (l. c.) divides the cases of mitral stenosis into three groups:

1. The congenital form, resulting from fetal endocarditis, which, as Sansom has shown, is very rare.

2. The endocarditic form of infectious origin (due especially to rheumatism, scarlet fever, measles, typhoid fever, smallpox, etc.), resulting from the organization and fusion of old vegetations. This is the most common form, since rheumatism alone can be demonstrated as an etiological factor in about 70 per cent. of the cases.

3. The sclerotic form, which is associated with general arteriosclerosis, gout, and plumbism. In this group no traces of endocarditic vegetations occur upon the valve, and the process is more closely allied to that within the vessel walls in arteriosclerosis. Under this group should also fall the large group of cases associated with chlorosis and other anæmias in which no other causal factor is present to explain the presence of the lesion. Goodhart, who lays especial stress upon this group, believes that the chronic overstrain of the anæmic heart muscle is followed by œdema and petechiæ in the substance of the mitral valve, as has been demonstrated experimentally by Roy and Adami. He thinks that this œdema is followed by cellular infiltration and finally by proliferation of fibrous tissue resulting in the mitral stenosis. While this view is suggestive, no careful histological or experimental studies have been made to bear it out. On the other hand, it must be remembered that most anæmic and gouty persons are subject to repeated slight infections which might suffice to produce chronic changes in

the heart during the lapse of years. Weber and Deguy have shown that hemorrhage occurs often in the valves after labor, etc. This is followed by infiltration and organization and finally by sclerosis of the valve.

PATHOLOGICAL PHYSIOLOGY.

The nature of the disturbance to the circulation in mitral stenosis was already discerned by Vieussens in 1715, who noted that owing to the inability of the blood to pass through the mitral orifice with sufficient rapidity, it had become dammed back in the pulmonary veins and pulmonary arteries, increasing the work of the right ventricle and leading to insufficiency of the tricuspid valve. At the same time the force of the pulse (blood-pressure) and amplitude of the pulse were diminished, owing to the diminution of the blood which entered or was forced out of the left ventricle. Vieussens noted further that the force and rhythm of contraction were irregular.

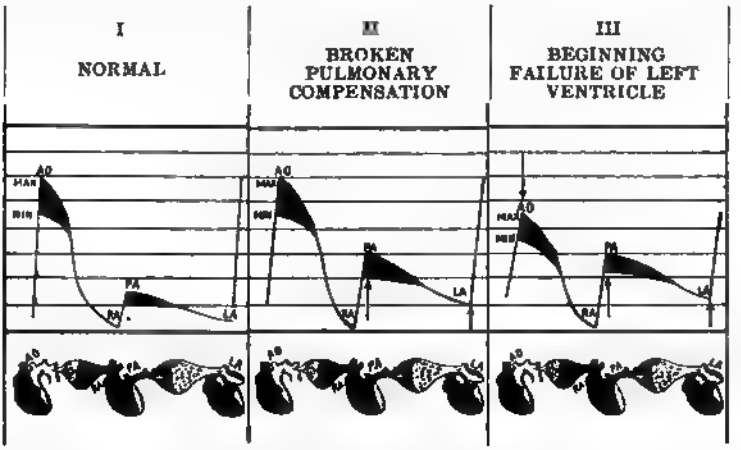

FIG. 190.—Diagram showing the changes in the circulation due to mitral stenosis. The stage of perfect compensation is not shown, since there are practically no pressure changes. II represents the stage at which pulmonary compensation is broken but systemic circulation has not yet become affected. III represents the stage in which there is broken pulmonary compensation, and the left ventricle has failed to maintain the arterial pressure. The last pressure changes in the last stage, with broken systemic compensation as well, correspond exactly to those in the last stage of mitral insufficiency.

The changes of pressure corresponding to these phenomena have been studied experimentally by Bettelheim and Kauders, D. Gerhardt, and MacCallum and McClure, who have found that the production of a mild grade of experimental stenosis causes distinct rise in the mean pressure within the left auricle, the pulmonary veins and the pulmonary artery.

Filling of the Ventricles.—The effect of the stenosis upon the filling of the ventricles as shown in the volume curve has recently been investigated by the writer, aided by Mr. J. M. Wolfsohn. As shown in Fig. 191, the first effect of the mitral stenosis is to slow the inflow into the left ventricle. As a result of this the left auricle is more than usually full at the time of its systole, and forces an unusually large quantity of blood into the ventricle (Fig. 192, AS). This increase in auricular output at first suffices to complete the filling of the ventricle, but as the lesion progresses a little further even this fails to do so and the amount of blood entering the ventricle falls below normal. As a consequence of this, the ventricle forces

less blood into the aorta, the arterial blood-pressure falls and the pulse-pressure diminishes. This is the condition as observed in the experiment. In man, however, where the pathological change is a gradual one, the arteries gradually accommodate themselves by constriction of their channels until the blood-pressure has returned to about its normal level. The pressure in the systemic veins is diminished, as has been shown by Kornfeld.

FIG. 191.—Volume of the ventricles in experimental mitral stenosis. Kindness of the Johns Hopkins Hospital Bulletin. Tracings obtained from a dog's heart in experimental mitral stenosis. *VOL*, volume curve. *CAR*, carotid pressure. The arrow indicates the moment at which the stenosis was produced. The filling of the ventricles (downstroke, also slowed diastasis, horizontal part of curve) sets in prematurely, and the part of the curve due to auricular systole (second downstroke) becomes more pronounced. The heart rate is practically unchanged.

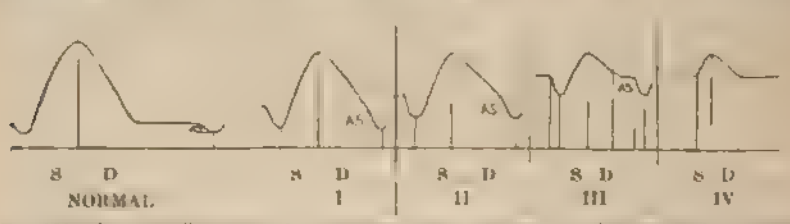

FIG. 192.—Diagram illustrating the variations in the volume curve of the ventricles in increasing degrees (I, II, III, IV) of mitral stenosis. *S*, systole; *D*, diastole; *AS*, inflow into the ventricles due to systole of the auricles.

As the narrowing of the mitral orifice progresses, a further change occurs in the filling of the heart. It now requires a greater difference in pressure to drive the blood from auricle to ventricle and as a consequence it is seen that during a great part of diastole little or no blood is flowing in. Diastole is thus divided, according to Henderson's terminology (cf. page 91), into three periods.

I. A period of early diastole, during which the ventricle fills with moderate rapidity, though more slowly than normally.

II. A period of diastasis, Henderson, during which little or no filling occurs. Experiments upon the excised heart (cf. page 9) indicate that the mitral valves close suddenly just at the beginning of this period and remain completely or almost completely closed until the next.

III. This is followed by the third event of diastole, when more blood is driven into the ventricle by the forcible contraction of the left auricle. The latter must now contract more forcibly than ever and as a consequence hypertrophies. Indeed Gerhardt has shown, from the autopsies of two cases with very mild grades of stenosis, that hypertrophy of the left auricle is the first change resulting from the lesion, and that hypertrophy of the right ventricle is a later phenomenon. Pulmonary congestion always occurs, however, and in all but the very mildest cases hypertrophy of the right ventricle results.

With further narrowing the filling of the left ventricle diminishes, its total volume and systolic output diminish, and the blood-pressure falls unless the constricting power of the vasomotor is taxed to the utmost to preserve it. The left auricle, on the other hand, becomes overloaded and distended, its capacity even rising from 60 c.c. to 2500 c.c. (G. Müller) or 3000 c.c. (Minkowski). Under these circumstances paralysis of the auricle sets in, the auricular wave disappears from the curve of filling (Hirschfelder), from the jugular pulse (Mackenzie), and the œsophageal tracing (Joachim), and the presystolic rumble is no longer heard. At this stage the rhythm of the heart usually becomes irregular, due no doubt to the effort of the auricle to empty itself by means of abnormal contractions. This stage of irregularity with auricular paralysis is more or less characteristic of cardiac overstrain in mitral stenosis, and may disappear when the work of the heart is relieved, or its strength is increased by digitalis (see Fig. 199), when the presystolic rumble reappears. As the lesion progresses further, the stasis in the pulmonary circulation increases, and broken compensation follows, just as it does in mitral insufficiency.

As can be seen from the above description, the problem of maintaining compensation in mitral stenosis is quite different from that in mitral insufficiency. In the latter condition the important factor was seen to be the emptying of the left ventricle, whereas in mitral stenosis the difficulty lies in the filling of this chamber, and the hypertrophy necessary to bring this about affects not the left but the right ventricle and the left auricle. Since this can be accomplished only by increasing the pulmonary engorgement, it is evident that after the lesion has reached a certain stage perfect compensation becomes impossible, and the re-establishment of compensation is always more difficult than in a corresponding grade of mitral insufficiency.

SYMPTOMS AND SIGNS.

The symptoms, color, and general appearance of patients with mitral stenosis are very much the same as in those with mitral insufficiency, except that they are somewhat more pronounced and more persistent. Hæmoptysis and precordial pain are more frequent than in mitral insufficiency, as are also attacks of pulmonary œdema.

Hofbauer, Alexander, Miecslaw, Frischauer, and Osler have called attention to the hoarseness and stenotic (brassy) cough as a symptom in mitral stenosis. Osler has shown at autopsy that under these conditions the left recurrent laryngeal nerve is compressed against the arch of the aorta and the ligamentum Botalli by the dilated left auricle, and then undergoes degenerative changes.

André Petit recognizes three clinical groups of persons with mitral stenosis:

I. The chlorotic type affecting mainly young girls. The skin and mucous membranes are pale, the complexion sallow. The patients readily become short of breath, are subject to nose-bleeds, menstrual disturbances, dyspepsia, constipation, and nervousness. Only auscultation reveals the nature of the disease.

II. The pseudotuberculous type in young women, characterized by repeated attacks of bronchitis, dry, hacking cough, and hæmoptysis, especially at menstrual

periods. The patients are pale and emaciated and closely resemble consumptives in appearance.

III. The dyspnoeic type into which any of the other types may merge. There is dyspnoea on exertion frequently also attacks of cardiac asthma at night. These patients usually have flushed faces with slight dull tinge of cyanosis (the typical mitral facies), due to imperfect aeration of the blood in the lungs.

PHYSICAL EXAMINATION.

Except for the findings in the immediate vicinity of the heart, the results of physical examination in cases of mitral stenosis are practically the same as in mitral insufficiency. The pulse, however, is rarely of as long volume as in the latter, and the pulse-pressure is usually smaller. Irregularity is about as common and is of the same type (absolute irregularity, with or without auricular paralysis) in both conditions.

The Cardiac Impulse. The findings about the heart are, however, characteristic. The apex impulse, sometimes in the normal position and sometimes out in the axilla, is usually well marked, sudden, short, and tapping or flapping. This peculiar appearance is due to the seesaw movements of the chest wall over the right and left ventricles. There is a wavy movement in the third, fourth, and fifth interspaces, which when accurately timed or recorded is found to consist of a systolic retraction of these interspaces over the hypertrophied right ventricle (see page 209). The systolic impulse of the pulmonary artery may sometimes be seen in the second left interspace at the sternal margin.

FIG. 193.—Diagram showing the direction of the stream entering the left ventricle through the stenotic mitral orifice.

Palpation. The findings of palpation are perfectly characteristic. At the apex, and usually over this area only, the shock of the **first sound can be felt, short, sharp, and tapping.** In the typical cases, up to the last stage (auricular paralysis) this is preceded by a short presystolic thrill, having a "purring" character (frémissement cataire, Corvisart, Laennec), leading up to the shock which accompanied the first sound. It can be increased by slight exercise. **It is, as a rule, sharply localized** about the apex in an area of about 3 cm. in diameter (or about the size of a stethoscope bell), probably because it is produced by the impact of the narrow stream driven by the forcible auricular contraction directly against the apical portion of the walls of the left ventricle.

Indeed Oestreich, Lenhartz, and Burk have shown that this stream may finally cause a bulging of the ventricular wall at the point where it has been striking. The vibrations transmitted from the chordæ tendineæ and papillary muscles are also factors in its production.

Occasionally a tap or thrill is also to be felt in early or mid-diastole accompanying the third heart sound or the diastolic rumbles about to be described. The sensation thus imparted by the presystolic thrill and the tapping first shock are so characteristic that the diagnosis

of mitral stenosis may often be established from palpation alone. The shock accompanying the second sound is usually very distinct, and becomes markedly accentuated as the pulmonary area is approached.

Percussion.—The typical area of dulness on percussion and the cardiac outline as seen by the X-ray (Groedel) often show the outline of the left ventricle (lower border and lower half of left border) to be normal, though in almost equal frequency it is extended to the left. Thus, in 32 cases Sansom found the left ventricle

Normal in	12
Hypertrophied in	9
Dilated in	5
Dilated and hypertrophied in	3
Small in	3
Thin walled in	1

Above, the area of dulness extends higher and further to the left than normal, often reaching as high as the second rib though usually not above the third and embracing an area of 2 to 5 cm. in the second left interspace.

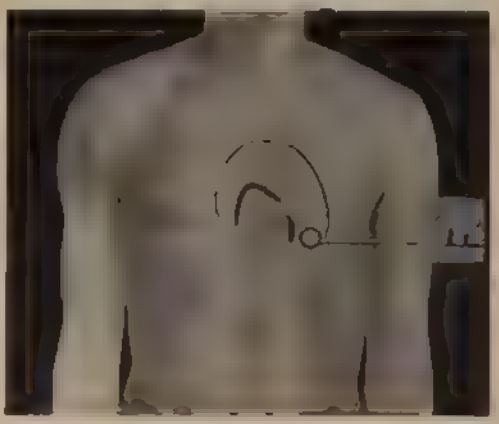

Fig. 194.—Cardiac outline and distribution of the presystolic murmur in mitral stenosis. [illegible] (Huchard).

As shown by the autopsies of Th. Harris this area of dulness corresponds to the dilated conus arteriosus and dilated pulmonary artery and not to the dilated left auricle. Except when general failure has set in, the right border of the heart is normal, but the cardiac flatness may be increased and reach to the right sternal margin—hypertrophy of the right ventricle. The typical area of cardiac dulness in mitral stenosis is therefore a short wide oval with a bulging upon its upper left margin (Fig. 194).

Auscultation.—**Presystolic Rumble.**—The auscultatory findings in mitral stenosis are quite unique, and usually furnish the basis for diagnosis. The characteristic sign is a rumbling or echoing sound in late diastole (auriculo-systolic in time), increasing in crescendo up to the beginning of the first heart sound. Like the presystolic thrill and the diastolic sounds, this presystolic rumble is usually heard over an area of 2–3 cm. only, at or a little to the right of the apex.

The rumble and the first sound thus come to have the phonetic equivalent of Tou-ter-rup Turrup (Steel Rou-tou Durouz). It was first described by Cendrin in 1842 and by Fauvel in 1845 in cases of mitral insufficiency, but its diagnostic significance was definitely established by W. T. Gairdner of Edinburgh in 1861. Gairdner and the older writers considered it to be produced by systole of the left auricle (auriculo-systole) driving the blood forcibly through the narrow mitral orifice. This view has been practically proved by the observations of James Mackenzie that the presystolic murmur is present only when the venous tracing shows contraction of the auricles and disappears when the auricle becomes paralyzed. Jonesium has also demonstrated this for the left auricle by œsophageal tracings. The writer has moreover been able to demonstrate this parallelism in experimental mitral stenosis. Einthoven's curves of the

heart sounds in man, as well as those of Weiss and Joachim, also prove the time of its occurrence. It is quite certain, therefore, that the rumble does not occur in the early part of ventricular systole, as claimed by Ormerod, Barclay, Turner, Dickinson, and Brockbank. The protodiastolic and mid-diastolic rumble remains, however, in spite of auricular paralysis.

Fig. 105.—Radiograph from a case of mitral stenosis, taken with the plate behind the patient, showing increase of the shadow due to the dilated left auricle. (Kindness of Prof. C. M. Cooper.) The left ventricle is not dilated.

Fig. 106.—Diagram representing the shadows shown in Fig. 105. The broken line indicates the outline of a normal heart. The arrow indicates the enlargement upward in the transverse oblique diameter. *LA*, left auricle, *PA*, pulmonary artery, *AO* aorta, *LV*, left ventricle.

Snapping First Sound. — The nature of the first heart sound is also characteristic. It is short and sharp, and may resemble the second sound so closely as to be mistaken for the latter. This is especially common in cases in which the second sound is not well heard at the apex.

Fenwick and Overend believed that it was of higher pitch than the normal first sound, but this is not borne out by the curves of Weiss and Joachim, who found waves of about normal pitch but of much greater amplitude (Fig. 197). It is possible that Fenwick and Overend may have heard higher overtones than accompany the normal heart sound. Owing to its loudness and sharpness, it may so closely resemble the normal second sound as to be mistaken for it. According to Hayecraft, it is the valvular element which imparts the high-pitched notes to the first sound, and thus is naturally accentuated in mitral stenosis, for the stiff mitral valve plays a greater rôle in sound production than in the normal.

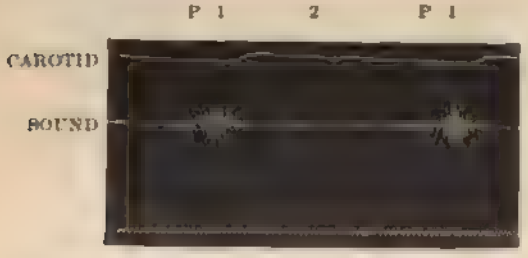

Fig. 107.—Graphic record of carotid pulse and heart sounds in mitral stenosis. (After Weiss and Joachim.) Upper line, carotid pulse; middle, cardiophonogram; lower line, time in fifths of seconds. *P* presystolic rumble, 1, 2 heart sounds.

Broadbent and Acland believed that the ventricular walls "close down rapidly because their cavity is not distended with blood" and thus give rise to a more sudden sound. However, the snapping sound is quite as pronounced in the numerous cases in which the ventricle is dilated, and moreover, on the other hand, the cardiometer shows that diminution in ventricular volume occurs at exactly the same rate as in the normal heart.

[1] On account of the smaller amount of blood discharged at the usual rate the duration of systole is shorter, but Einthoven's tracings show that the duration of the first sound is normal.

This applies only to the first sound produced in the left ventricle, and hence heard only over a small area about the apex from which it is transmitted for a short distance upward and to the right. Over the right ventricle the first sound is normal until the latter stages of the disease, when it is often replaced by the murmur of tricuspid insufficiency. The second sound at the apex is usually faint, and may even become quite inaudible in the latter stages of the disease, probably owing to the low arterial blood-pressure, but over the pulmonic area it is markedly accentuated.

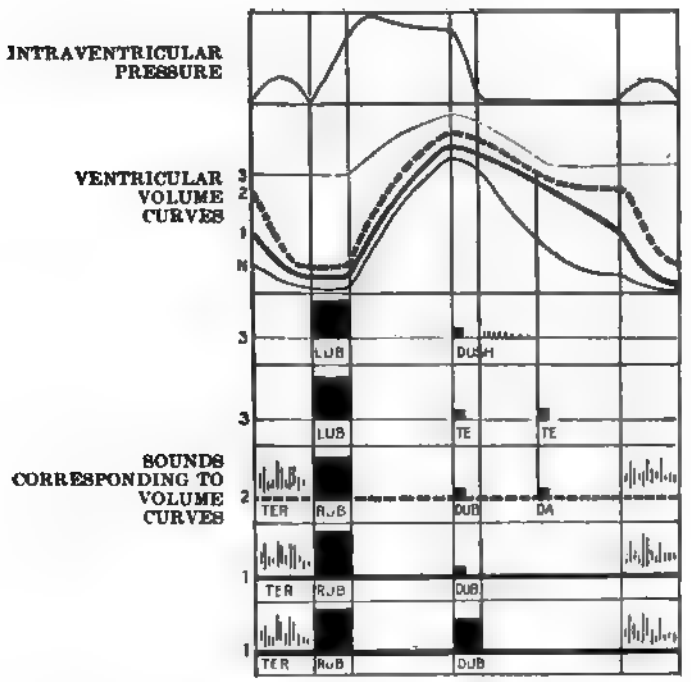

FIG. 198.—Diagram showing the relations of the various sounds heard in uncomplicated mitral stenosis to events in the filling and emptying of the ventricle. *N*, normal. Light, heavy, broken, and dotted lines correspond with sounds whose base lines are made with these lines.

Digital Imitation of the Presystolic Rumble and Snapping First Sound.—While the ordinary vowel and consonant sounds fail to give any close imitation of the sounds heard in mitral stenosis, these may be quite closely reproduced by the method of H. L Smith (page 115), tapping on the knuckles with four fingers in rapid succession.

Systolic Murmur in Mitral Stenosis with Mitral Insufficiency.—Since the mitral cusps are much deformed and thickened in mitral stenosis, and the chordæ tendineæ shortened, it is not surprising that leakage at this valve should be present in the greater number of cases (Fig. 179). The group of signs due to the insufficiency of the valve associated with stenosis do not present striking differences from the signs of the former lesion when existing alone. Hypertrophy of the left ventricle occurs in almost all these cases, and the heart is enlarged horizontally to the left. A blowing systolic murmur accompanies and follows the snapping first sound; and occasionally, when the second sound at the apex is absent or very soft, this murmur may seem to be diastolic Careful timing by comparison with the shocks and the time of the pulmonic second sound, however, will prevent this error.

Sounds in Early and Mid-diastole.—The sounds heard in early and mid-diastole, however, are quite as well marked as is the presystolic murmur. These sounds are of three types:

1. An *early diastolic* sound, the *third sound* of a proto-diastolic gallop rhythm (rup - tat - ta), which Bouillaud observed in 1841 in association with mitral stenosis. This sound is heard in about 65 per cent. of all cases of mitral stenosis (Stovell). Though somewhat louder it does not seem to differ from the third sound of the normal heart in mode of production, and is probably due to the sharp closure of the mitral cusps when the period of rapid ventricular filling has come to an end. It is therefore to be regarded as a "closing snap" of the mitral (Hirschfelder, l. c.), and not as an "opening snap" as thought by Duroziez, Sansom, and Potain. As the filling of the ventricle is somewhat less complete than normal, this sound occurs a trifle early (Thayer). That it should be louder and more readily produced is due to the rigidity of the altered mitral valve.

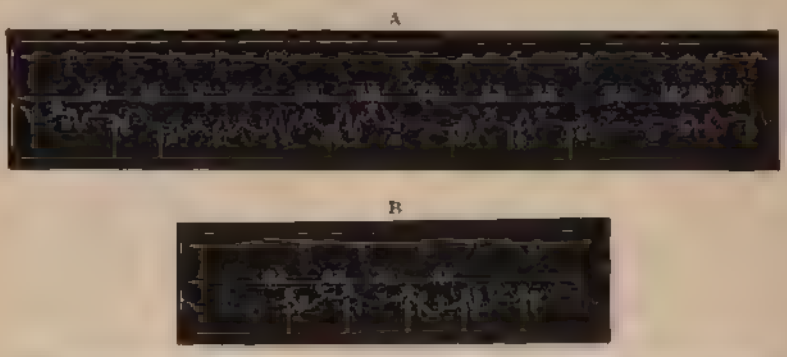

FIG. 190.—Venous pulse of a patient with mitral stenosis during an attack of acute heart failure. A, Tracing taken on Dec. 1, 1907. The presystolic rumble has disappeared. The *a* auricular wave is absent from the venous pulse. There is absolute arrhythmia. B, Tracing taken five days later after return of compensation. The presystolic rumble has returned. The *a* wave is present on the venous pulse. The rhythm is regular. The arrows mark the time when auricular contraction should occur.

2. One of the commonest sounds in mitral stenosis is a *rumble in early or in mid-diastole* of the same character as the presystolic rumble. According to Huchard, it may resemble the sound of a flag fluttering in the breeze, causing the cardiac cycle to resemble the words "rup - tat - rarou." This rumble may follow almost immediately after the second sound. It may last throughout diastole and be continuous with the presystolic rumble, or it may be separated from the second sound and also from the presystolic by short intervals. On the other hand, when the presystolic rumble has disappeared during auricular paralysis it may be the only sound heard during diastole.

These rumbles occur, therefore, at the time when the blood is flowing rapidly into the left ventricle. At this phase of diastole, owing to the stasis in the pulmonary veins, the auricular pressure is relatively high. It is therefore not surprising that the stream passing through the narrowed auricular channel under about the same pressure should give rise to a rumble similar to that produced later in diastole when the same stream is propelled through the same orifice at about the same rate by the force of the auricular contraction. Nor is it, therefore, surprising that, as shown by Mackenzie, the early diastolic

rumbles produced by a pressure which is continually diminishing should be always decrescendo in character, while the presystolic rumbles produced by the rapidly increasing auricular contraction should, on the contrary, be crescendo.

3. A blowing diastolic murmur not unlike the ordinary murmur of aortic insufficiency is occasionally heard in mitral stenosis (Graham Steell, Huchard, Cabot and Locke, Bard). This murmur, however, never replaces the second sound as does the aortic murmur, but follows it after a short pause. It becomes louder and lasts longer as the pulse-rate diminishes. The murmur is intense at the apex, but is also heard above and to the left of the latter. It is not heard over the aortic area nor over the sternum, though sometimes heard at the upper left sternal margin. Whether this murmur is caused by a secondary functional insufficiency of the pulmonary valves, due to dilatation of the right ventricle (cf. page 390), or is a cardiopulmonary murmur (cf. page 111) induced by the increased activity of the right ventricle, is still unsettled. It is probable that each is met with in some cases.

STAGES OF MITRAL STENOSIS.

It is difficult to divide the course of mitral stenosis into well-defined stages. From a physiological and diagnostic stand-point it might be convenient to distinguish four stages,—which differ slightly from those described by Broadbent (l. c.). These are characterized by the following phenomena:

First Stage. — Presystolic rumble, snapping first sound and second sound are well heard at the apex,—compensation is good,—the left auricle contracting forcibly as shown by the presystolic rumble, also the left ventricle as shown by well-marked second sound. The second pulmonic sound is increased. The sounds of early and mid-diastole may or may not be present. Broadbent says of this stage, "I have never known serious symptoms to arise from the condition of the heart, and I have seen serious illnesses of different kinds passed through without the intervention of embarrassment of the circulation."

Second Stage.—The presystolic rumble and first sound persist and the early and mid-diastolic rumbles may be present, but the second sound at the apex has completely or almost completely disappeared. This indicates that the force of the left ventricle is beginning to diminish (Fig. 190, III), but the left auricle is still contracting forcibly. At this stage the presystolic rumble is sometimes mistaken for the first heart sound and the snapping first sound for the second, so that if there is a systolic murmur present a diagnosis of mitral insufficiency may be made, and a less severe prognosis is sometimes given. This error may be avoided by carefully timing the heart sounds by palpation or by noting the change in sounds on gradually passing the stethoscope from base to apex.

Third Stage.—The presystolic rumble and thrill have disappeared. The snapping first sound and sounds of early diastole persist; the second sound at the apex may or may not be heard. Paralysis of the left auricle has set in.

Overlapping of Second and Third Stages.—In some cases this occurs earlier than the disappearance of the first sound at the apex, and these two stages overlap or may even replace one another chronologically.

This depends upon the relative strength of left ventricle and left auricle, as well as upon the degree to which the mitral orifice is narrowed; for a comparatively weak left auricle sometimes fails without materially affecting the cardiac filling, while, on the other hand, a vigorously beating auricle may produce a loud sound while forcing a small amount of blood through the narrowed orifice, and yet the amount of blood thus entering the left ventricle may be too small to maintain the arterial blood-pressure and to cause a distinct second sound.

Dyspnœa, hæmoptysis, and the usual features of stasis in the pulmonary circulation occur in the second and third stages of the disease.

Fourth Stage.—Broken systemic compensation, venous stasis, œdema of the extremities, enlargement of the liver, gastric and digestive disturbances, ascites, hydrothorax, and all the other features of broken compensation of the right ventricle set in. In the later stages there are well-marked signs of tricuspid insufficiency, positive venous pulse, and positive pulsation of the liver.

PULSE.

Exactly as in mitral insufficiency, permanent irregularity of the pulse due to the numerous extrasystoles may occur quite early in cases where both the presence of the presystolic rumble and the venous pulse tracing

Fig. 200.—Permanent arrhythmia in a case of mitral stenosis, showing persistence of the auricular contractions, a wave, upon the venous pulse. The right auricle, at least, is still contracting. The tracing shows the presence of persistent auricular extrasystoles.

show that the auricles are still contracting forcibly (Fig. 200). Owing to the greater circulatory disturbance entailed in the filling of the left ventricle, arrhythmia causes a somewhat greater circulatory disturbance in mitral stenosis than in insufficiency, although in neither is it a harbinger of immediate danger and it may last for years. The pulse is usually small in mitral stenosis, owing to the contracted condition of the radial and other medium-sized arteries.

The blood-pressure is, as a rule, very little changed, owing to the compensatory changes in the arterial bed. The most common change is a diminution in pulse-pressure due to a rise in the minimal pressure brought about by the vasoconstriction. A large pulse-pressure, such as is frequently seen in well-compensated mitral insufficiency, is not common in mitral stenosis.

DIAGNOSIS.

In the absence of aortic insufficiency and adherent pericardium the diagnosis of mitral stenosis is comparatively simple, and is based upon the presence of the presystolic or diastolic rumble or thrill at the apex, together with a short snapping first sound in this region, and a markedly accentuated second pulmonic sound.

However, it sometimes happens that these signs appear only occasionally while the patient is under observation.

Some years ago the writer had under his care for several months a patient with pernicious anæmia in whom a presystolic murmur was heard on only one occasion though the heart's action was fairly vigorous and regular. A gallop rhythm had been present during her entire illness. The diagnosis of a mild grade of mitral stenosis was verified at autopsy.

Occasionally, on the other hand, the patient is seen after auricular paralysis has set in and when there is no diastolic rumble present whatever and only the sounds of the second and third stages. Such cases show the need of frequent auscultation of the patient.

Differentiation between Aortic Insufficiency and Mitral Stenosis.—In the presence of aortic insufficiency it must be remembered that the presystolic and diastolic murmurs described by Austin Flint may closely simulate those arising in mitral stenosis (see page 371). In aortic insufficiency the thrill is rarely as well marked as in mitral stenosis, and the first sound is rarely sufficiently short and snapping to be mistaken for mitral stenosis. The two conditions were found together in 1.2 per cent. of the 1781 cases of valvular disease at the Johns Hopkins Hospital,—i.e., in about one-seventh of all the cases of mitral stenosis and one-tenth of all the cases of aortic insufficiency, so that it is not a condition of extreme rarity. When, as not infrequently happens, the mitral stenosis is the first condition present, there is no difficulty, as the late diastolic blow of mitral stenosis is rare and scarcely ever mistaken for aortic insufficiency. But when the aortic insufficiency is the first lesion to occur, the diagnosis of the second lesion becomes much more difficult. Occasionally in doubtful cases a slight exercise or a few forced expirations with glottis closed (Valsalva's experiment) will increase the work of the left auricle and cause the presystolic thrill and rumble to become so intense that the presence of mitral stenosis is unmistakable. A diagnosis should never be made unless the heart has been examined in various stages of its activity. In spite of such precautions errors are not infrequent, and are made by the most skilful observers. It must be frankly confessed that there are many cases in which the diagnosis cannot be made with any degree of certainty.

According to Phear, adhesive pericarditis can also be mistaken for mitral stenosis, since occasionally a presystolic rumble may occur, due no doubt to the stretching of the fibrous strands under the influence of the auricular contraction of the ventricular filling, and here also the diagnosis of two coexistent lesions should be made with caution.

Presystolic Gallop Rhythm.—Another condition which on rare occasions may be confounded with mitral stenosis is one of slight cardiac weakness in which there is a presystolic gallop rhythm. Under these circumstances, as Sewall states, the ventricles are overfilled with blood and the auricle encounters some difficulty in forcing blood into them. There may even in some cases be a slight functional stenosis like that discussed on page 106. Sewall believes that under these conditions the contraction of the auricle becomes audible and may even be mistaken for mitral stenosis.

Quite recently the writer has had under his care in the Johns Hopkins Dispensary a girl of 13 years who presented this picture. She had had a slight attack of rheumatism and tonsilitis and shortly afterwards began to have palpitation, weakness, nervousness,

and a little pain in the precordium and around the lower part of the left axilla. On examination the heart was not enlarged, but the shock accompanying the first sound was quite sharp and there was a well-defined impulse which began with vibrations that suggested a presystolic thrill. These were somewhat increased on exercise. The second pulmonic was not abnormally accentuated. The pulse-rate was 120 and regular. There was no anæmia. The signs were not quite definite enough to warrant a diagnosis of mitral stenosis. The patient was given digitalis for a few days and this was followed by a prolonged treatment with iron, quinine, and strychnine. She improved steadily and for several weeks past no presystolic sounds or impulse can be elicited even by quite severe exercise. As it seems quite unlikely that an auricular paralysis would set in coincident with these conditions of improvement, and in the absence of any arrhythmia, it seems most likely that this case represented one of very loud presystolic gallop rhythm, and that no organic lesion is present.

Case of Mitral Stenosis with Insufficiency, Angina Pectoris, and Pulmonary Sclerosis.

O. A. K., farmer, aged 34, was admitted to the Johns Hopkins Hospital, June 2, 1903, complaining of heart trouble and shortness of breath. Family history negative. Patient has always been healthy except for chorea at 14 and acute articular rheumatism at 18. He is rather subject to headaches. Has smoked and drunk in excess, but for the past few years does so in great moderation. He is much exposed to the weather.

For the past six or seven years he has been short of breath and has had pain over the heart. Six months before admission he developed ascending oedema, which disappeared under treatment, but his breath remained short and the oedema reappeared within a couple of months. During the past month he has had spells of dizziness and fainting during exertion and had one chill followed by fever.

On examination the patient is seen to be a well-nourished man with anxious appearance and flushed face, deeply cyanotic. He is quite dyspnoeic. The teeth are bad, the pharynx is injected. The veins of the neck are prominent, but do not pulsate. The chest is full, and coarse râles are everywhere heard.

Heart. The apex impulse is seen in the 6th left interspace 12 cm. from the midline. Dulness extends upward to the second left interspace and 4 cm. to the right of the sternum. There is a loud systolic murmur heard over the entire heart and left axilla. The heart's action is irregular in force and rhythm. Pulse-rate 76. The radial is somewhat sclerotic. Maximal blood-pressure 130 mm. Hg.

His liver and spleen were not enlarged. There was no oedema of the feet.

He was kept in bed, purged freely, given tincture of digitalis (8 doses of 1 cc. [mxv] each). During the following two months his condition improved markedly, in spite of the occurrence of a fibrinous pleurisy. His dyspnœa diminished and he felt much improved. Coincident with this improvement a presystolic rumble and thrill gradually appeared and a snapping first sound preceded the systolic murmur. Three months after admission his cyanosis had almost entirely disappeared. The heart was still enlarged (the apex 11 cm. from the midline), a well-marked presystolic thrill was felt and a presystolic murmur was heard at the apex. The first sound was snapping and was accompanied and followed by a loud blowing systolic murmur.

The blood-pressure during his stay in the hospital ranged between 120 and 135 mm. Hg.

After leaving the hospital at this time the patient felt well for about six weeks, during which period he could even run for a car without distress. Then he caught a cold which persisted for four months, being aggravated by exertion. He had two more fainting spells on exertion. Oedema returned, the lower extremities and of late even the face and eyes being swollen. Urine less frequent than normal.

On readmission he was very cyanotic and very dyspnoeic. Moist râles were heard over the entire chest. The apex is now in the 6th left interspace 15.5 cm. from the midline. The presystolic rumble and systolic murmur are well heard, as at the last discharge. Red blood-corpuscles 5,000,000, hæmoglobin 105 per cent., leucocytes 5500.

On the night of admission the patient felt badly and had attacks during which he felt faint and "saw stars." Venesection caused much relief in the symptoms and the blood-pressure rose from 120 mm. Hg to 140 mm. The patient's condition then gradually improved, but on January 24 he had a definite attack of precordial pain and constriction lasting 1–3 minutes.

Feb. 23. Has had pain in head and the left side of the face due to a beginning otitis media.

During the next few weeks he had several attacks of angina pectoris, the pain being usually most marked behind the lower part of the sternum. In one attack it radiated to the left shoulder and down the left arm to the hand (left ventricular pain).

On April 20 he complained of pain in the left axilla and back, coming in paroxysms lasting for 15–20 minutes (left auricular pain).

May 6. Sputum bloody. From this time on he gradually improved, cyanosis and dyspnœa almost disappeared, and he was discharged in August in fair condition. From that time until his readmission in November he suffered from numerous attacks of angina pectoris, beginning in the left hand and passing up the arm to the shoulder and heart. He also had an attack of rheumatism and severe tonsillitis. His physical condition was like that on previous admission, but the anginal attacks were more frequent. He was given hypodermic injections, sometimes of morphine, sometimes of distilled water, to relieve them. His condition gradually improved, most markedly after venesection. Toward the end of his stay, while up and about, he became subject to sudden paroxysms of acute dyspnœa (respirations 130 per minute), with small moist râles filling the lungs (acute pulmonary œdema). These persisted in spite of repeated rest and digitalis treatments. He was discharged on July 11, 1905, seven months after admission. Œdema and dyspnœa returned within ten days, and he was soon back in the hospital again. During this admission he never thoroughly rallied. His blood-pressure remained low, 105 mm. maximal pressure (as compared with 120–130 mm. on previous admissions). The pulse was irregular. Râles were constantly present in his chest. The liver was palpable. A slight pleurisy developed on October 13 and he died on October 20.

At autopsy the mitral orifice was found to have the form of a small button-hole barely admitting the tip of the little finger (about 5 mm. in diameter). The left auricle was dilated and hypertrophied. The left ventricle was not dilated, but was much hypertrophied, its walls being 15 mm. thick. The right auricle and ventricle were much dilated, the tricuspid orifice admitting four fingers (13–14 cm. in circumference). The coronary arteries were patent but showed scattered areas of sclerosis. There were old fibrous patches upon the pericardium. The pulmonary arteries were markedly sclerotic; the sclerosis extended into their smaller branches. The aorta and peripheral arteries showed less sclerosis. There were also chronic passive congestion of the other viscera, anæmic infarctions of the spleen, hemorrhagic infarction of the lungs, acute bronchitis, bronchopneumonia, acute ulcerative follicular colitis, old tuberculous foci in the lymph-glands and lungs, chronic adhesive pleuritis, and adhesive peritonitis.

COMPLICATIONS.

As seen by the table in Fig. 179 mitral stenosis is frequently associated with other valvular lesions. Mitral insufficiency, present in one-half of the Johns Hopkins cases and in 75 per cent. of Steell's cases, may be regarded as an essential part of the disease rather than a special complication, and its presence does not shorten the average length of life.

The association of aortic disease, and indeed of any additional burden upon the circulation, increases the gravity of the condition.

Tricuspid stenosis is an occasional concomitant, though rarely as often found as by Samways, who encountered it in severe grade in 24 out of 196

autopsies upon cases of mitral stenosis, and in mild grade in 8 additional cases. In the Johns Hopkins Hospital it was found clinically 7 times among 298 cases of mitral stenosis

A certain degree of myocarditis is the rule, especially in cases in the third and fourth decades.

Acute pericarditis is quite common in the youthful rheumatic cases, frequently leaving a residual adherent pericardium, a lesion which aggravates the condition considerably and greatly shortens the life of the patient.

One of the most important and dangerous complications arising with mitral stenosis is pregnancy (see Chapter IX).

Thrombosis in the Left Auricle.—Another not uncommon complication of mitral stenosis, more than any other valvular lesion, is thrombosis within the left auricle. This may occur even while the auricle is still contracting vigorously, as shown by the presystolic thrill and rumble. The thrombus may remain quiescent in the tip of the auricle or it may obstruct the pulmonary veins. Sometimes it is so large as to stop up the narrow mitral orifice and kill the patient. More frequently it is small enough to pass through, and if carried on by the blood current gives rise to a small area of embolism.

Embolism.—Embolism of the middle cerebral artery may give rise to paralysis or aphasia. Embolism in other organs gives rise to corresponding signs and symptoms.

Pulmonary embolism and infarction are caused by thrombi from the right auricle and ventricle; and hence are due to secondary stasis in the latter and not primarily to the mitral stenosis However, as failure of the right ventricle is particularly common in mitral stenosis, pulmonary embolism is especially frequent in this disease.

A few months ago a patient was admitted to the medical service of the Johns Hopkins Hospital with gangrene and anæsthesia of both lower extremities, due to plugging of the abdominal aorta by such an embolus He had a well-marked mitral stenosis with purring presystolic thrill and loud presystolic rumble, showing that his left auricle was contracting vigorously. Needless to say, nothing could be done to relieve him, and he died within a few days.

TREATMENT.

The treatment of mitral stenosis is practically the same as that of mitral insufficiency, except that, since the lesion is a more uniformly progressive one, greater care must be exercised in the general hygiene, especially the avoidance of infection and overstrain. Digitalis is given about as in mitral insufficiency, especially when the left ventricle is dilated

Amyl nitrite and the other drugs of the nitrite group may prove of value when there is a broken pulmonary compensation. Hydrotherapy and gymnastics may be used after the pulmonary compensation has been re-established, but must be administered with even greater care than in mitral insufficiency, for acute pulmonary engorgement and pulmonary œdema are more liable to set in. Since it is particularly important to guard against dilatation and hypertrophy of the right ventricle, venesection should be performed promptly in failure of the latter, especially if there are signs of pulmonary œdema, unless marked anæmia is present.

Hypodermic injections of atropine 0.5–1.0 mg. (gr. $\frac{1}{120}$–$\frac{1}{60}$) may be given to relieve the pulmonary œdema or to ward it off.

Owing to the rôle of anæmia in the etiology of mitral stenosis, it is especially important that the hæmoglobin be kept at a normal level. If anæmic, the patient should be given as complete rest as possible, with maximal amount of fresh air and sunshine, a diet especially rich in eggs, on account of the lecithin, and iron preparations, especially Blaud's pills (0.3 Gm., gr. v), or Vallet's mass (same dose), or Ferrum oxidatum saccharatum solubile (one teaspoonful in water), should be administered three times a day. When the hæmoglobin is near 60 per cent. or the progress slow, arsenic should be given by the mouth, especially as Fowler's or Donovan's solution, in increasing doses until 1 c.c. (15 minims) is reached. Prof. J. O. Hirschfelder obtains excellent results by the hypodermic injection of 1 per cent. sodium arsenate (1 c.c., 15 minims) daily. However, this must sometimes be discontinued on account of pain, in which case atoxyl may be substituted.

W. Arbuthnot Lane and later (1902) Sir Lauder Brunton suggested that in the light of modern surgical technic it might be possible to slit the narrowed valve with a fine knife and thus remove the stenosis. The experiment of slitting the mitral valve has been performed by Cushing and Branch in hearts of normal dogs. It does not present extreme difficulties, but the recoveries were few, in spite of the fact that the heart muscle of these dogs was in good condition. Bernheim in the same laboratory arrived at similar results. Lauder Brunton had advocated the operation only for cases which were otherwise doomed; and it is evident that here the danger from a weakened myocardium would be far greater. Moreover, even if successful, the mechanical effect of suddenly converting a severe mitral stenosis into a severe mitral insufficiency would impose an intense strain upon the heart, and might, even in that way, do more harm than good.

PROGNOSIS.

In spite of the numerous complications and the progressive character of the lesion, the average duration of life in mitral stenosis is not extremely short, being 33 years for males, 38 for females. This is due to the large number of cases in which the process is dormant or progresses very slowly, and indicates in the individual case the importance of avoiding everything which may start it afresh, particularly infections and overstrain. In many cases the condition then remains dormant for many years, the patient continuing to live a normal if somewhat careful life without further trouble. Lenhartz has seen cases pass through seven pregnancies without manifesting signs of cardiac distress, and endurance of equal magnitude may be met with in men. On the other hand, the lesion may progress rapidly and death may occur within a few years. In the more severe cases it may come on without warning, often due to the loosening of an embolus from the left auricle.

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IV.

AORTIC INSUFFICIENCY.

HISTORICAL.

Our knowledge concerning lesions of the aortic valves producing leakage at that orifice (aortic insufficiency, aortic regurgitation, aortic incompetency) dates from 1705, when the English anatomist Cowper first described the occurrence of stiffening and thickening of the valves so that they "did not apply adequately to each other, whence it happened sometimes that the blood in the great artery would recoil and interrupt the heart in its systole." Shortly after this, and quite independently of Cowper, Vieussens (1715) described two similar cases, noting also the presence of a very full quick pulse, like the rebound of a tightly-stretched cord, associated with palpitation of the heart so severe that it prevented lying down. Morgagni also described several cases, in one of which he recognized both aortic insufficiency and aortic stenosis. Hodgkin in 1829 described a number of cases, and also noted for the first time the diastolic murmur, but did not recognize any diagnostic features; so that the clear clinical picture of aortic insufficiency may be said to date from the publication of Dominic Corrigan in 1832

FIG. 201.—Specimen showing vegetations upon the aortic valves. The arrow points to the vegetation

PATHOLOGICAL ANATOMY.

Modern classifications of aortic insufficiency differ little from that of Corrigan, and we distinguish as he did—

1. Organic forms of aortic insufficiency due to pathological changes in the valves.
2. Functional or relative aortic insufficiency due to dilatation of the mouth of the aorta (Fig. 202, D).

The organic forms of aortic insufficiency may be of three types:

1. Endocarditic, due to the occurrence of inflammatory changes upon the valves, usually vegetations, occasionally to calcified atheromatous plaques (Fig. 202, A).

2. Rupture of the valves, sometimes from mechanical strain, sometimes from ulceration (Fig. 202, B).
3. Sclerotic shrivelling of the cusps, usually associated with arteriosclerosis (Fig. 202, C).

1. The pathology of the endocarditic lesions has been sufficiently discussed in a preceding chapter (page 299), since they represent quite typical vegetations. This form of lesion results from the usual causes of endocarditis, especially rheumatism, scarlet fever, pneumonia, as well as gonorrhœal, puerperal, septicæmic, and other acute infections (see page 301). It is the most common form in persons below thirty-five, whereas the sclerotic is more common in later life.

2. Rupture of the valves is one of the less frequent but by no means rare occurrences, and usually takes place suddenly during a period of great muscular strain, such as wrestling, lifting a heavy weight, drawing a heavy burden, or even during a bicycle race (Huchard), or else after severe blows upon the chest (Osler). Under these circumstances, as has been seen, the blood-pressure may suddenly rise to a tremendous height (see page 132),

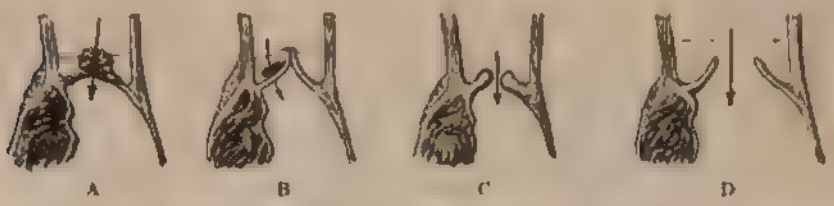

FIG. 202.—Schematic showing the various forms of lesion producing aortic insufficiency. A. Vegetation. B. Perforation. C. Arteriosclerotic shrinking. D. Dilatation of the aorta.

and the blood stream tears its way through the valve at the weakest point—usually near the base of the sinus of Valsalva. The ruptured valve may have an apparently normal structure, but probably contains minute areas of degeneration, since it is impossible to rupture a normal valve experimentally by subjecting it to the highest pressures that are ever reached in the animal body. In many cases the ruptured valves show arteriosclerotic changes which have tended to weaken the tissue. Where endocarditic changes are already present, rupture or perforation of the valve takes place spontaneously and at ordinary or even lowered blood-pressures.

3. The sclerotic form of the lesion is not only the most common, but produces the lesions which are most uniformly severe and progressive, since it usually occurs in later life and is associated with general arteriosclerosis (Fig. 202, C). The lesion is in every way similar to the sclerotic process elsewhere in the aorta, and may assume any of the types which occur in general arteriosclerosis—thickening with contraction, calcification, aneurismal dilatation, perforation. In any individual case the symptoms depend upon the size of the leak as well as upon the state of the peripheral vessels and the heart.

The etiological factors bringing about this lesion are the same as those given for general arteriosclerosis (see page 254), infections, alcohol, tobacco, lead poisoning, nephritis, hard work. Osler (l. c.) especially calls attention to the fact that syphilis is one of the most frequent causes of sclerosis

about the root of the aorta in men under thirty-five, and that the lesions commonly involve both the aortic valves and the mouths of the coronary arteries. He considers this factor next to endocarditis in order of importance as a cause of aortic insufficiency in young men. Collins and Sachs and Longcope have recently obtained a positive Wassermann reaction in about half the cases of aortic insufficiency in which there had been no outspoken rheumatism.

Sex.—In women aortic insufficiency is far less frequent than in men, constituting 8.4 per cent. of all heart lesions in the former as compared with 28.5 per cent. in the latter (Gillespie).

Moreover, as shown by Romberg and Hasenfeld, the presence of aortic insufficiency from causes other than sclerosis in itself leads to the production of general arteriosclerosis, and hence the presence of any other form of the lesion predisposes to the superposition of sclerosis.

Functional Aortic Insufficiency.—The existence of leaks at a dilated aortic orifice was already suspected by Corrigan, especially when there was an aneurism near the base of the ascending arch. This has been verified by subsequent observers and a diffusely dilated aorta with insufficiency of the valves is not a rare finding. As regards the presence of transitory leaks from dilatation Gibson has also shown experimentally that such a dilatation may occur as a result of too high pressure in the excised heart, and Stewart claims to have produced it by cutting the aortic ring muscle. But since transitory aortic insufficiency does not often accompany the high blood-pressures of uræmia, meningitis, and brain tumor, it is probable that this factor plays little rôle clinically. The cases of supposed functional aortic insufficiency are rare, but Anders has reported a considerable number. In some at least it is possible that the phenomena (diastolic murmur, collapsing pulse, etc.) are due to other causes, especially functional pulmonary insufficiency. Cardiopulmonary murmurs, like those described by Potain, must also be excluded. However, the possibility of functional aortic insufficiency must be borne in mind by the clinician, but it can rarely be verified, and the clinical diagnosis is, at best, hazardous.

Fig. 203.—Effect of aortic insufficiency in the mechanical model. (After Marey.) The horizontal line shows the point of production of aortic insufficiency. *PV* intraventricular pressure. *PK* arterial pressure. *O* auricular systole. The diastolic pressure in the ventricle after aortic insufficiency is considerably higher than in the normal condition and approximates the diastolic pressure in the aorta. The wave-let *a* due to contraction of the auricle, is less marked.

PATHOLOGICAL PHYSIOLOGY.

The mechanical effects of aortic insufficiency upon the circulation were first studied experimentally in horses and dogs, as well as on the mechanical model by Marey and Chauveau.

They produced the lesion by pushing a probe down one carotid artery and through a cusp of the aortic valve, while they registered the pulsation in the other carotid by means of a cannula. They were thus able to reproduce the phenomena observed clinically, especially the occurrence of the murmurs, the violent beating of the heart and arteries, and the large collapsing pulse which had been described by Corrigan. They were also able to reproduce these phenomena in a mechanical model of the circulatory system.

The experiments of Marey and Chauveau on animals have been repeated and confirmed by Cohnheim, Rosenbach, de Jaager, Kornfeld, Romberg and Hasenfeld, and those upon the model by Moritz. The subject was again investigated under the writer's direction by Dr. H. A. Stewart in the Johns Hopkins Medical Clinic. The method employed by Stewart differed from that of previous observers in the fact that he recorded simultaneously the volume of the ventricles, the maximal and minimal blood-pressures, and the pulse-curve from the carotid artery. He found in animals, as had been shown by Marey upon the mechanical model, that the production of aortic insufficiency is at

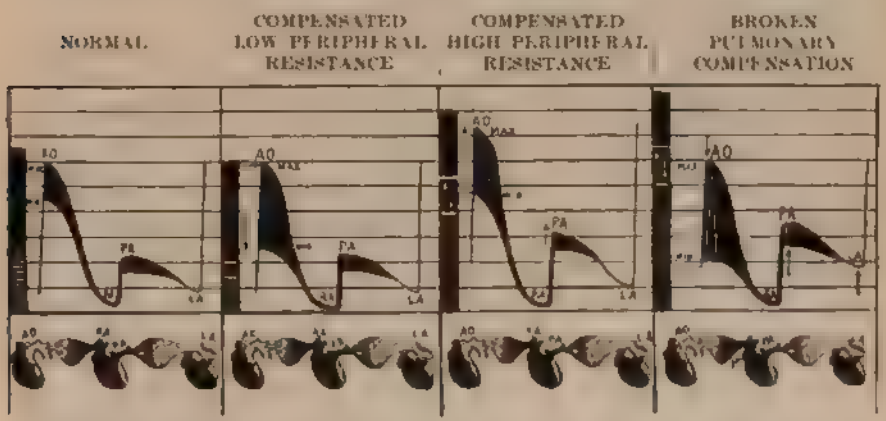

FIG. 204. Diagram of the circulation in aortic insufficiency. The vertical black bars represent the volume of the left ventricle, the shaded portions representing the residual blood, the portions indicated by the white arrows showing the amount of blood regurgitating, the solid black indicating the systolic output. The black arrows indicate the change in condition or pressure that has taken place. MAX, MIN, maximal and minimal blood pressures. The white curve indicates the absolute sphygmogram corresponding. AO, PA, RA, LA as in previous diagrams.

once followed by a great fall in pressure during diastole, which is at least in part, due to the regurgitation into the ventricle. This great fall in diastolic pressure is the most characteristic feature of aortic insufficiency.

The actual amount of blood regurgitating, both in animals (Stewart) and in model experiments (Moritz) is usually not more than one tenth of the total forced out at each systole.

As in the case of flow through any orifice, the factors influencing this regurgitation are: (1) the size of the hole in or between the valves; (2) the head of pressure in the aorta; (3) the length of time during which leakage occurs.

Cardiac Tonicity. The experiments performed by Stewart and the writer indicate that the chief factor antagonizing the reflux is the elasticity of the heart muscle, or the cardiac tonicity.

It is evident that, with a given lesion and a constant heart rate, the factor affecting the regurgitation is, therefore, the antagonism between the height of blood-pressure during diastole, on the one hand, and the cardiac tonicity, on the other. However, the pressure within the ventricle is not

constant throughout diastole, but is continually increasing; while the pressure in the aorta is continually decreasing, and the reflux will cease altogether at the instant when pressure within the ventricle + cardiac tonicity − pressure within the aorta. Consequently, the lower the pressure in the aorta (diastolic pressure) or the higher the cardiac tonicity, the earlier this will occur and the less will be the amount of blood regurgitating.

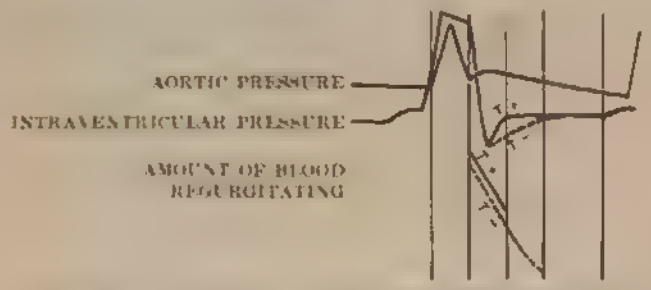

FIG. 205.—Diagram showing how the high cardiac tonicity (T +) hastens the equilibrium between aortic pressure, intraventricular pressure, and tonicity, and thus diminishes the amount of blood regurgitating.

It was found, however, that whenever this occurred the hearts dilated and the animals died. Those animals which survived the shock of the operation were able to increase their systolic output by the amount regurgitated, and thus in spite of the lesion to keep the maximal pressure at the same height as before. In these animals positive intraventricular pressure during diastole acts as a load to the heart muscle, which responds

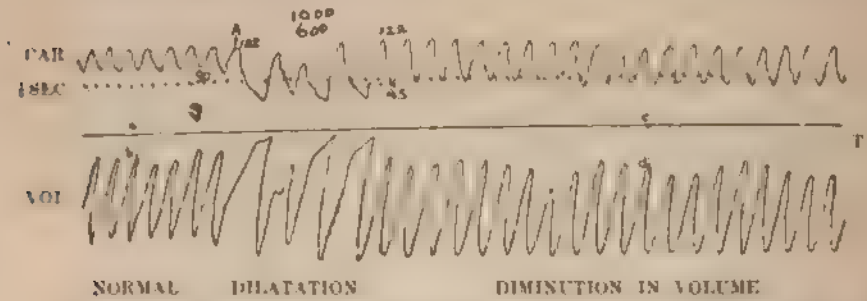

FIG. 206.—Effect of rupturing an aortic valve in a dog showing a transitory dilatation followed by a permanent diminution in size. After Stewart. *CAR*, cardiac; *VOL*, volume of the ventricles. Downstrokes represent systolic upstrokes diastole. The maximal blood pressure remains unchanged 122 mm. Hg; the minimal pressure falls from 90 to 45 mm. Systolic output is somewhat increased. *a-b* represents the tonicity of the ventricles before producing the lesion, *c-d* represents tonicity after the lesion. *A* represents the point at which the aortic valve was ruptured.

by increased tonicity, and fills somewhat less completely than it otherwise would; so that the total volume of the ventricles after aortic insufficiency, just as after any other strain, may be smaller than before it. In these hearts total volume is decreased, systolic output increased, and residual blood greatly decreased.

Romberg and Hasenfeld (l. c.) have denied that this increased tonicity is always beneficial, claiming that by inhibiting the inflow from the auricle it impedes the circulation. However, Kornfeld found that the pressure in the left auricle is not affected by experi-

mental aortic insufficiency as long as the strength of the left ventricle remains unimpaired. As the influences which maintain tonicity are in almost every instance the same as those which increase the strength and volume of the systole, it is probable that Romberg and Hasenfeld are in error.

As regards the rôle played by tonicity in aortic insufficiency two views are held:

1. Romberg and Hasenfeld claim that an increased tonicity hindering the influx of blood from the auricle is distinctly harmful.

2. Stewart and the writer have shown that the most dangerous event in experimental aortic insufficiency is overdilation, and this is antagonized by increase in tonicity. Moreover, Cloetta has found that the hearts of rabbits with experimental aortic insufficiency which had been treated with digitalis were less dilated and were much stronger than those of normal rabbits. Almost all the influences which bring about increased systolic output are the same as produce increase in tonicity. Fear of evil results from this cause seems, therefore, quite unwarranted.

Blood-pressure.—As regards blood-pressure, it was found that when the peripheral resistance was increased, as by clamping the thoracic aorta, the force of the heart-beat increased correspondingly, and both maximal and minimal (systolic and diastolic) pressures increased about equally and pulse-pressure remained high. With the increase in diastolic pressure upon clamping, the regurgitation through the orifice increased (Fig. 214), and, as systolic output changed no further, the heart dilated considerably, showing that high peripheral pressure represents the condition which produces the greatest embarrassment of the circulation. With the increase in peripheral pressure, however, the form of the pulse curve changed from collapsing to flat-topped and anacrotic, a fact which will be referred to later.

Rate.—Corrigan thought that if the heart were slowed and diastole were prolonged the heart would undergo great dilatation, but in the experiments performed by Stewart and the writer it can be seen that this dilatation soon reaches its limit, and the volume of the ventricles need not exceed the normal volume for the same rate (Fig. 206). This is due to the fact that, as the aortic pressure falls during a prolonged diastole, it approaches the intraventricular pressure, and the above-mentioned equilibrium is soon reached.

As a result of experiments upon animals, it would appear, therefore, that the conditions most favorable to the heart are low peripheral resistance and moderately high tonicity; and, as will appear later, therapy should be directed toward this end.

Pulmonary Circulation.—As stated above, Kornfeld's experiments show that the aortic lesion has no effect upon the pulmonary circulation as long as the left ventricle is acting powerfully. When the left ventricle begins to fail, pulmonary stasis and rise in auricular pressure occur (Kornfeld), which, as Stewart and the writer have found, are frequently associated with the occurrence of a functional mitral insufficiency. Pulmonary stasis in aortic insufficiency is, therefore, a secondary phenomenon dependent upon failure of the left ventricle. The clinical importance of this fact will be referred to subsequently.

Hypertrophy.—As a result of the increased strain upon the left ventricle, the walls of this chamber undergo great hypertrophy. The cavity of the left ventricle, owing to the regurgitation during diastole, is often much dilated, especially in the infrapapillary or aortic portion of the chamber.

The left auricle, on the other hand, is rarely hypertrophied. The right ventricle, however, usually shows some hypertrophy, resulting either from slight increase in pulmonary pressure or from continuity of the fibres with those of the left ventricle.

SYMPTOMS.

The symptomatology of aortic insufficiency differs considerably from that of the mitral lesions. When the lesion is well compensated and no pulmonary stasis occurs, dyspnœa may not appear for years, and in the meantime the patient may enjoy excellent health. On the other hand, he may also be considerably annoyed by the throbbing of his arteries, headache, roaring in the ears, by loss of memory, by periods of depression often alternating with periods of great exhilaration, by the appearance of motes or muscæ volitantes before the eyes.

Hallucinations of sight, especially that of the veiled gray figure, of hearing (rhythmic knocking or bell-tolling), and of smell are relatively common in aortic disease, and are usually associated with pain in the precordium or down the arms and tenderness over the upper left chest (Head). Head states that these always disappear when mitral insufficiency sets in.

There are often pains over the heart, especially over the base, and down the left arm, and typical attacks of definite angina pectoris. These symptoms are especially common in the sclerotic forms, in the later stages of the disease, but may occur even when the coronary arteries are unaffected. After the break in compensation, dyspnœa is usually intense, and the patient is compelled to sit up in bed, not only on account of shortness of breath but also on account of extreme palpitation.

Cheyne-Stokes respiration (of the cardiac type) is somewhat more common in aortic insufficiency than in other forms of cardiac disease. Undoubtedly this is due to some disturbance in the medullary circulation, but the exact explanation is still uncertain.

Sudden agonizing attacks of cardiac asthma are very frequent, accompanied by intense orthopnœa, in which the patient gasps for breath for several minutes or even half an hour. No doubt they are associated with sudden overfilling of the left ventricle, secondary functional mitral insufficiency, and pulmonary stasis. Sudden death may occur during such attacks, and is indeed more common in aortic insufficiency than in other forms of valvular disease. Both the maximal and the minimal pressures may be high up to the instant of death, as in one case observed by the writer in which the blood-pressures were 150 mm. Hg and 110 mm. respectively until the instant when the pulse suddenly ceased.

PHYSICAL EXAMINATION.

The appearance of persons suffering from aortic insufficiency is usually quite characteristic. The eyes are bright, with conjunctivæ moist, the pupils often dilated and palpebral slits wide, giving a peculiar staring ap-

pearance which sometimes for an instant suggests exophthalmic goitre. The scleræ are usually pale and bluish. The cheeks are somewhat sunken, the complexion usually pale and sallow (aortic facies).

One of the features which at once attracts the attention of the observer, as already the case with Vieussens in 1715, is the intense and sudden visible pulsation in the arteries, especially the carotids, temporals, and brachials. Often this pulsation is so great as to shake the entire head, even when the patient is asleep (Fränkel). There is sometimes a visible pulsation of the entire uvula (F. Müller) and of the arteries in the retina (Becker).

Not only the larger but also the smaller arteries pulsate visibly, as can be seen in the so-called capillary pulse (Quincke), the to-and-fro movement of the red border of an area of hyperæmic skin synchronous with each pulse-wave. This can be observed at any place where an area of erythema borders upon an area of pallor, especially along the margin of an area of skin which has been caused to redden by slight friction, beneath the finger-nails, or in the lips or gums when gently compressed with a glass slide. This appearance coupled with the presence of the bounding and collapsing pulse is usually very typical. Indeed, it is said that Oppolzer won his professorship at Vienna by casually making a diagnosis of aortic insufficiency while walking down the wards of the hospital and merely resting his hand upon the dorsum of the patient's foot.

However, Oppolzer might readily have come to grief had he encountered one of those not very rare cases in which all these phenomena result from arteriosclerosis alone. Lennhoff, v. Weissmayer, and Huber have termed these cases pseudo-aortic insufficiency. In these cases the arteries are large and rigid and there is a high pulse-pressure but no other manifestations of aortic insufficiency. The whole phenomenon is due to a high pulse-pressure in rigid arteries (page 261).

The chest and lungs show no abnormalities until the later stages of the disease are reached and pulmonary congestion has set in with bronchitis, pulmonary œdema, or hydrothorax.

Cardiac Impulse.—Over the heart there is usually some bulging of the chest wall, and usually a well-defined apex beat in the fifth or sixth interspace to the left of the mammillary line. The impulse is systolic in time and heaving in character (dome-like, "choc en dome"), owing to the fact that the entire apex is usually made up of the hypertrophied left ventricle (Bamberger). In the second right interspace there is often another systolic impulse, caused by the throbbing aorta, which may lead to the suspicion of aneurism.

On palpation nothing abnormal is noted except that the second aortic shock is often lacking. In about 40–50 per cent. of the cases a presystolic thrill (Thayer) and in 15 per cent. a tapping systolic shock may be felt at the apex. This is very similar to that observed in mitral stenosis but less intense, while in most cases the impulse is strong and heaving. In about 16 per cent. of Thayer's cases an actual mitral stenosis was present as well, and this association must always be borne in mind. Systolic thrills are often felt, especially over the aortic area, caused by the roughenings of the aortic valves, and also over the apex in cases where mitral insufficiency is present.

Percussion and X-ray Shadow.—As stated above, the cardiac outline in aortic insufficiency shows a marked elongation of the long axis (L), due to the hypertrophy and dilatation of the left ventricle. There is little increase in the transverse diameter (Q), so that the area of cardiac dulness

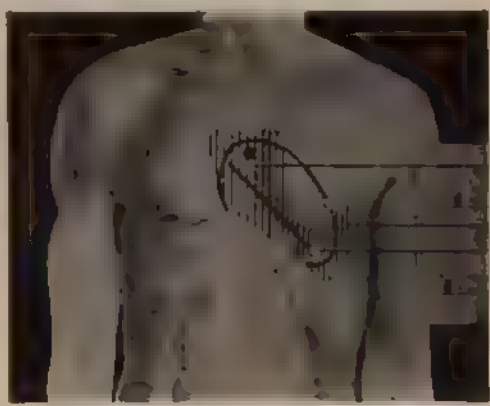

FIG. 207.—Area of cardiac dulness and distribution of the cardiac sounds and murmurs in aortic insufficiency. Heavy curved line, outline of the heart; heavy straight line, longitudinal diameter. Parallel lines indicate the distribution of the aortic diastolic murmur. Black dot indicates maximum intensity. Small circle indicates the distribution of the presystolic rumble (Flint). Small diagram at the left indicates the murmur heard in each area.

and the X-ray show (Figs. 207, 208, and 209) the form of a narrow elongated oval whose long axis is inclined more obliquely downward than is that of the normal heart. In this way it presents a marked antithesis to the out-

FIG. 208.—Radiograph of a case of aortic insufficiency showing elongation of the long axis of the heart. (Kindness of Prof. C. M. Cooper.) The plate is at the back of the patient, the tube in front.

FIG. 209.—Diagram of Fig. 208 showing the hypertrophy of the left ventricle. The broken line indicates the normal cardiac outline; the arrow indicates the direction of enlargement; AO indicates the shadow of the dilated aortic arch.

line of mitral stenosis, in which the oval is a broad and rather short one, and to the broad, elongated oval of mitral insufficiency. The X-ray shadow often shows a marked dilatation of the aortic arch, which may correspond to an area of dulness in the second right interspace and over the adjacent portions of the sternum, but this can be differentiated from aneurism by

fluoroscopic examination with oblique illumination. Indeed, examination with the X-ray shows this condition to be much more frequent than had previously been suspected, and discloses many cases of dilated aorta which had previously been regarded as true aneurisms. On the other hand, the tremendous strain upon the vessel walls in aortic insufficiency tends to bring about the formation of aneurisms, and the latter is a relatively frequent complication of aortic insufficiency.

The Aortic Diastolic Murmur.—The characteristic and almost pathognomonic sign of aortic insufficiency is the blowing, hissing, or occasionally musical murmur heard over the heart in early diastole. This murmur was first described by Hodgkin in 1829 as "a constant bruit de scie, which presented this peculiarity, that it was double, attending the systole as well as the diastole."[1] However, it remained for Corrigan (1832) to recognize its diagnostic significance. The murmur is caused by the regurgitant stream passing through the orifice between the closed valves, and its quality, like the noise made by a jet of steam, depends upon the size and character of the opening and the pressure in the vessel during diastole rather than upon the size of the orifice. Indeed, a small leak passing through a narrow orifice, especially with irregular and calcified walls, at a high diastolic pressure, may cause a much more intense murmur than a large leak through a wide orifice (cf. page 110). Balfour even goes so far as to state that when the diastolic murmur is loud over the base but not over the carotid artery the regurgitation is small, whereas when it is loud over the arteries but not heard over the base the leak is a large one. In occasional cases of ulcerative endocarditis separation of an entire cusp may occur without the presence of the characteristic murmur. Moreover, it is frequently observed that the diastolic murmur is totally absent when the heart is rapid and weak, but reappears as the rate falls and the force of the beat increases. The consistency of the valves also plays a considerable rôle. A rigid and calcified orifice forms a better sounding-board and gives rise to a louder and more roaring murmur.

As to the region in which it is best heard, the statements of different authors vary. The following list shows the region of maximum intensity given by various authors:

V. Jurgenson	Second right interspace and adjacent portions of sternum.
Gerhardt	Left of sternum.
Romberg	Second and third left interspaces.
Huchard	Third right costal cartilage.
Sibson	Lower part of left margin of sternum.
Broadbent	Sternum near origin of third left costal cartilage.
Osler	Midsternum, third costal cartilage, or along left border of sternum as low as ensiform.

Cole and Cecil have called attention to the fact that in many cases of aortic insufficiency the diastolic murmur not only can be heard but undergoes an accentuation as the stethoscope passes outward from the apex into the left axilla. The writer can confirm this observation.

[1] Evidently there was a systolic as well as a diastolic murmur present.

The discrepancies in the statements of the different observers may be due to the direction taken by the regurgitant stream. Foster, Balfour, and Grocco suggested that this might depend upon the aortic segment which happened to be affected. It is easy for any one to demonstrate to his own satisfaction that this view is at least partially correct. A calf's heart may be obtained from a butcher's shop and a cannula connected with a pressure bottle inserted into the aorta. A window is cut into the left ventricle and a hole is then made in one of the aortic leaflets. A regurgitant stream issues from the hole taking a direction nearly perpendicular to the plane of the valve. The stream emerging from a hole in the left cusp strikes the septum, that from the posterior cusp strikes the left wall of the ventricle in the vicinity of the apex or anterior papillary muscle, while that from the right cusp strikes against the anterior cusp of the mitral valve. The higher the pressure under which these streams pass the more their direction is deflected toward the apex. A moderate change in pressure will make a great difference in the direction taken by the stream.

I II III

Fig. 210.—Direction of the primary regurgitant streams in aortic insufficiency. Schematic. I. Regurgitant streams passing through orifices in the aortic cusps. II. 1, Direction taken by a stream regurgitating at low pressure. 2, direction of stream regurgitating through the same orifice at high pressure. III. *A*, direction naturally taken by a stream regurgitating through an orifice in the aortic cusp. *B*, direction to which the stream through the same orifice is deflected by irregularities upon the surface of the vegetation.

If the orifice from which the stream emerges is an irregular one like that at the margin of a vegetation, the direction of the stream may be totally deflected from its original course (Fig. 210) and this is probably the case in most clinical conditions. No hard-and-fast rules hold for all lesions of any individual segment. However the important fact is that the regurgitant stream continues as a well-defined jet whose sound would naturally be loudest near the point where it strikes and which would be transmitted more or less nearly in the direction of its course. The walls of the heart and the chordæ tendineæ aid in transmitting these murmurs for some distance beyond their point of impact.

It is evident, therefore, that the mere variations in the direction of the regurgitant streams due to the form of the leak, the blood-pressure, and the position of the heart may give rise to the greatest variations in the point at which the murmur is maximal, and may account for the discrepancies in the clinical findings of excellent observers.

Murmurs over the Arteries. Owing to the roughening of the aortic valves and sometimes to the presence of aortic stenosis, a systolic murmur is also heard over the aortic area and transmitted along the blood stream to the arteries.

In the carotid and brachial arteries a diastolic or to-and-fro murmur may also be heard. This was described by Corrigan in 1832 and by Da Costa Alvarenga in 1856, but it is most frequently and easily heard over the femoral arteries, where it was first noticed by Bouillaud and described by his pupil, Duroziez, in 1861. It is usually known as Duroziez's double murmur. The diastolic portion is probably due to a slight regurgitant stream from the periphery toward the larger arteries.

Flint's Presystolic Rumble.—Another and very important murmur is the presystolic rumble heard only at the apex (Flint murmur), first described by Austin Flint in 1862 in cases of aortic insufficiency without any mitral involvement. This rumble is in every respect similar to that of mitral stenosis, and it is extremely difficult to determine whether the latter is absent.

Indeed Flint, in his original paper, supposed that the murmur was due to the existence of a functional narrowing of the orifice between the mitral cusps, which, as Baumgarten (1843) and Hammernjk had shown was closed at the beginning of auricular systole. Guiteras and Thayer believe that the murmur is due to the vibration of the anterior cusp of the mitral valve set in motion by the regurgitant stream. Thayer and also Gibson deny the existence of such a functional stenosis. However, the writer has been able to show on the excised heart, by the method of Baumgarten and Gad, that although the mitral valve usually opens along its entire extent yet when the pressure within the ventricle is increased the separation of the cusps occurs at only a small portion of the line of closure (Fig. 212).

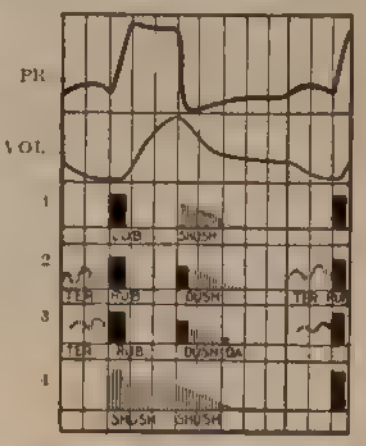

FIG. 211. Relation of murmurs in aortic insufficiency to the cardiac cycle. *PR*, intraventricular pressure; *VOL*, volume curve of the ventricles. 1, simple aortic diastolic murmur. 2, Flint presystolic rumble and aortic diastolic murmur. 3, presystolic rumble diastolic murmur and third sound. 4, mitral and aortic insufficiency murmurs replacing both sounds (machinery murmur).

An actual functional stenosis is, therefore, present exactly as assumed by Flint.

Snapping First Sound.—The first sound at the apex in 30 per cent. of these cases has the snapping character present in mitral stenosis, but more commonly is loud and booming. The systolic murmur transmitted to the left axilla is present in many cases in which mitral insufficiency is associated.

Aortic Second Sound.—When there is great destruction or great retraction of the aortic cusps and they do not approximate, the second sound may completely disappear, but if the edges are sclerotic or calcified, or covered with hard vegetations, the closure may even cause an intensification of the second sound, in spite of the presence of a larger regurgitation.

FIG. 212. Functional mitral stenosis in aortic insufficiency as demonstrated on the excised heart by Baumgarten's method. Semi-schematic.

Third Heart Sound.—Besides these sounds Prof. Thayer has called attention to the extreme frequency of a loud third heart sound (**protodiastolic gallop rhythm**) **in aortic insufficiency**, associated with the protodiastolic wave upon the cardiogram. If, as has been suggested by Hirschfelder, Gibson, and Thayer, this sound is due to the closing snap in diastole, it is quite natural that it should be unusually loud in aortic insufficiency when the mitral valves are forcibly clapped together by the high diastolic pressure in the ventricle. It can be readily shown on excised hearts that the snap is then more abrupt than under normal conditions.

BLOOD-PRESSURE.

The blood-pressure in patients with aortic insufficiency presents the same characteristics as in experimental animals,—namely, a constantly high pulse-pressure. This may be due either to a considerable fall in the minimal pressure (as, for example, maximal pressure 120, minimal 50), as is most common in the endocarditic group, or to a considerable rise in the maximal pressure with relatively little change in the minimal (170 and 90 respectively), such as is usual in the arteriosclerotic form. Occasionally one encounters cases in which an aortic diastolic murmur and normal pulse-rate are present with normal maximal and minimal pressures (120 and 90), but all the experimental evidence indicates that in these cases the leak must be a small one, just as is the case in animal experiments when a thrombotic deposit plugs the hole in a punctured valve (see page 299). In such cases, therefore, there is a definite lesion of the aortic valves, producing but little leakage yet a well-marked murmur. Any disturbances to the circulation in such a case are due to sclerotic and myocardial factors rather than to the aortic lesion. Although this class of cases has not been studied extensively, it seems probable that a careful functional diagnosis based upon the blood-pressure findings might prove very useful for prognosis.

FIG. 213.—Variations in the form of the pulse-wave encountered clinically in aortic insufficiency. (After Mares.) 1, normal form; 2, collapsing (Corrigan); 3, 4, 5, 6, anacrotic pulse.

PULSE.

The typical pulse of aortic insufficiency is very characteristic, and since the time of Corrigan has been known as the Corrigan pulse (water-hammer pulse, see page 47). The wave is large, with a quick upstroke (pulsus celer et altus) and sudden fall, leaving the artery quite

small and soft during diastole (collapsing pulse).[1] In the typical sphygmogram these characteristics are very marked. The criterion for designating a pulse-tracing as collapsing is not the steepness of the up-and-down strokes, for these depend chiefly upon the speed at which the smoked surface is travelling, but lies in the fact that the dicrotic notch in the collapsing pulse falls below the middle of the pulse-wave, while in the normal and anacrotic pulse it lies above the middle (Mackenzie and Broadbent). Since Marey and Huerthle have shown that the systolic period occupies the time before the dicrotic notch and the

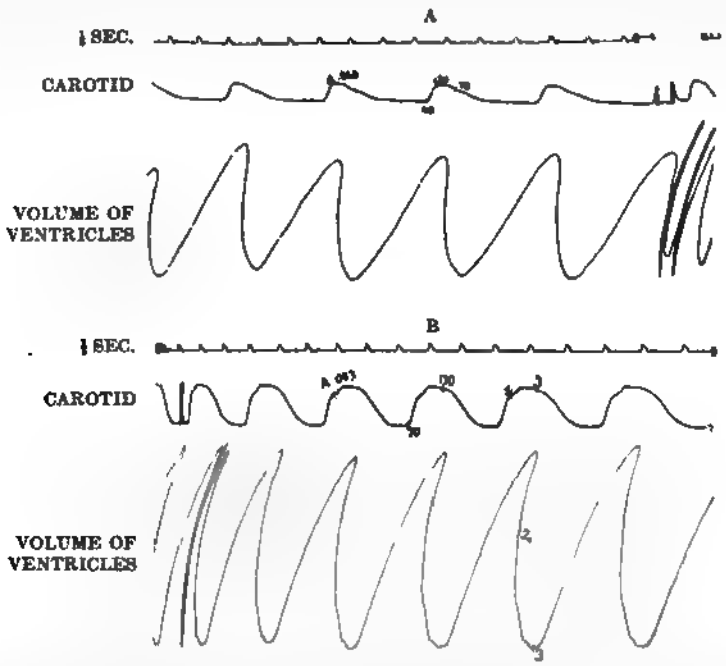

FIG. 214.—Tracings from a dog with experimental aortic insufficiency, showing the conversion of a collapsing (A) into an anacrotic pulse (B) by clamping the descending aorta. (After Stewart.) A, before; B, after clamping. The figures on the first pulse curves indicate the time from the base to the summit of the pulse-wave. The figures on the second wave indicate maximal and minimal pressures in the carotid. Downstrokes upon the volume curve represent systole. Clamping the aorta causes the ventricle to dilate somewhat, and to fill more quickly in diastole.

diastolic after it, it is but a paraphrase to state that in the collapsing pulse over half the fall of pressure occurs during systole, while in the normal pulse the fall occurs chiefly during diastole. Moreover the volume curves by Stewart and the writer have shown that the collapse occurs while the blood is still flowing out of the ventricle into the aorta, and not at the time when the regurgitation is occurring. On the other hand, increasing the peripheral resistance, either by clamping the descending aorta or by the administration of adrenalin, etc., which

[1] Corrigan called attention to the fact that elevating the arm caused the pulse to have a more collapsing quality, but Stewart has shown that this is due to hastening the venous return and not to increased regurgitation. It can be prevented by slightly constricting the arm.

caused an actual increase in the amount of blood regurgitated, caused the collapsing form of the pulse-wave to be replaced by one of typically anacrotic form (Fig. 214). The pulse-pressure, however, remained high. Moreover, the pulse-tracings of Marey (Fig. 213) showed almost all possible variations of form to occur in cases of aortic insufficiency, and Stewart found that the collapsing pulse was absent in 42 per cent. of the tracings at the Johns Hopkins Hospital.

There is also a certain number of cases in which the clinical note describes the pulse as collapsing, while no such character appears on the tracing. In these cases there is usually a large pulse-pressure, and the discrepancy is due to the fact that the finger appreciates the amount of the changes in pressure while the sphygmograph records mainly the suddenness of the change.

The cause of the collapsing character of the pulse seems, therefore, to be situated in the peripheral arteries, though the relatively small backflow into the ventricles and the high intraventricular pressure during diastole also play important rôles.

Hasenfeld and Romberg have shown that these vessels become greatly dilated after the lesion has been produced, and Stewart has shown that this is due to a stimulation of the depressor nerve at the aortic ring which the increase in intraventricular pressure may render continuous. Eastman has found by measurements of skin temperature that in typical aortic insufficiency the peripheral vessels are actually dilated. The blood therefore passes rapidly into the small arteries, and the aorta empties itself rapidly, so that this factor coupled with the backflow into the ventricle causes the great fall in pressure during diastole.

It is sometimes thought that the absence of a collapsing pulse in a case of aortic insufficiency indicates the presence of aortic stenosis. However, as not only the above-mentioned experiments but many autopsy findings demonstrate, this is not the case. It merely indicates that there is high peripheral resistance, which is common in arteriosclerosis.

Fig. 215.—Radial pulse tracings from patient (R. C.) showing extrasystoles A, which are probably of ventricular origin. The heavily shaded portion represents the systolic period of the cardiac cycle. The pulse is collapsing.

Pulse-rate.—The pulse-rate depends largely upon the degree of compensation, being little faster than normal in cases without symptoms, but usually ranging from 80 to 120 in hospital cases. The pulse-rate is usually regular; but when the heart's action is very labored, ventricular extrasystoles may result from the over-distention, and thus produce an irregular or a bigeminal pulse.

Case of Aortic Insufficiency

R. M. C., colored laborer, aged 16, entered the hospital on May 20, 1904, complaining of soreness and swelling of the abdomen and shortness of breath. He has always been healthy except for measles, mumps, and whooping-cough as a child, diphtheria at 21, and chills and fever. He has had gonorrhœa but denies syphilis. He passes water once a night. He has worked hard, exposed to wet and cold and doing heavy lifting. Does not use alcohol or tobacco.

About two years before admission he had cough, palpitation, shortness of breath on exertion, and some orthopnœa, of which he was cured at the dispensary. The present trouble began about two months ago, with violent beating of the heart and shortness of breath which were worse at night. These symptoms came on in paroxysms, which may be produced by stooping down. He has severe paroxysmal coughing spells. During the past few days his abdomen has been swollen, but his feet have not been at all so.

The patient is a poorly nourished man, propped up in bed without respiratory distress. There is marked pulsation visible in all the large arteries, and a to-and-fro murmur can be elicited over them by heavy pressure with the stethoscope. The lungs are clear on percussion and auscultation, except for a few coarse mucous râles at both bases.

Heart.—There is considerable pulsation over the precordium, the apex being located in the seventh left interspace 18 cm. from the midline. Dulness extends upward to the second left interspace and 5 cm. to the right of the midline in the fourth interspace. There is a well-defined presystolic thrill over the apex. A blowing systolic murmur replaces the first sound and is heard over the whole axilla, also a short blowing diastolic murmur and a short presystolic rumble (Flint murmur). Passing inward and upward the sounds are replaced by a loud to-and-fro murmur, loudest over the insertion of the fourth rib and scarcely heard to the right of the sternum. In the second right interspace the systolic murmur is very harsh and the first sound is loud; the diastolic murmur is well heard. The pulse is of good volume, regular, markedly water-hammer in character. Maximal blood-pressure 170 mm. Hg.

Abdomen is full; the hepatic dulness extends 9 cm. below the costal margin; the liver is soft and tender. There is well-marked œdema of the legs.

Red blood-corpuscles 5,000,000; hæmoglobin 75 per cent.; leucocytes 75 per cent.

He was put at rest, soft diet, and given 1 c.c. of tincture of digitalis and strychnine, 1.5 mg. (gr. $\frac{1}{40}$) every four hours, and purged freely. He immediately improved. The œdema disappeared, and within three weeks he was up and about, feeling well, with a pulse-rate of 80–90 per minute. He was discharged on June 16. His shortness of breath and œdema returned, however, within a week, and he re-entered the hospital on June 26 so dyspnœic that he was scarcely able to speak. The signs were about as before. He did not improve as before, however, in spite of treatment, and remained dyspnœic throughout the month, subject to spells of intense cardiac asthma, so that he was compelled to seek relief by being propped up continually in a wheel-chair instead of lying in bed. During this period his maximal pressure remained high (150–170), his minimal pressure about 110 (Erlanger apparatus). During the afternoon of August 1 the cardiac asthma was particularly intense. His maximal pressure was 150 mm. Hg, the minimal 110. Venesection was contemplated. Before this could be done, however, while the patient was being examined and perfectly conscious, the maximal blood-pressure suddenly fell to 110, the pulse stopped suddenly, and the patient dropped back dead.

At autopsy the aortic segments were found to be thickened at their edges, and the valve was clearly incompetent. The aortic orifice measured 8.5 cm. in circumference, the pulmonic 8.5 cm. The edge of the mitral valve was slightly thickened, the tricuspid also; but there was no stenosis of either. The tricuspid orifice, on the contrary, measured 14 cm. There was much dilatation and hypertrophy of the left ventricle, the heart weighing 760 Gm. The coronary arteries were clear. There were numerous thrombi along the walls of the right ventricle. Corresponding to this finding there were numerous areas of embolism of the branches of the pulmonary arteries, evidently from the loosening of such thrombi. Along the pericardium there were numerous raised irregular pearly fibrous patches. There were chronic passive congestion and œdema of the lungs, right-sided hydrothorax, chronic passive congestion of the abdominal viscera, and chronic gastritis.

DIAGNOSIS.

The diagnosis of aortic insufficiency usually presents little difficulty. The following conditions may, however, give rise to blowing diastolic murmurs over and near the sternum which may be mistaken for aortic insufficiency (Cabot and Locke).

1. Dilatation of the aorta.
2. Intense anæmia.
3. Tuberculous mediastinitis and similar conditions (murmur is cardio-respiratory, loudest in inspiration).
4. In association with mitral disease and dilatation of the pulmonary artery (functional pulmonic insufficiency)

These conditions are, as a rule, easily excluded, and do not frequently obscure the diagnosis.

On the other hand, as has been seen, it is often difficult to exclude complications such as mitral stenosis in the presence of a well-marked Flint murmur, or of aortic stenosis when the pulse is not collapsing.

The dilatation of the aorta, which often follows as a sequela of aortic insufficiency, is frequently mistaken for aneurism. It may be accompanied by very marked pulsation in the second right interspace and even of the upper portion of the sternum, with dulness in these regions. Even the X-ray when taken in the anteroposterior diameter may be ambiguous, and oblique illumination may be necessary to remove the suspicion of aneurism (Holzknecht).

The existence of a functional aortic insufficiency from transitory dilatation of the aortic ring can only be definitely diagnosed when an aortic diastolic murmur and an abnormally high pulse-pressure have been present and have passed off. This is indeed a rare occurrence. Other complications are few, and are generally those of arteriosclerosis. Bronchial features may be present, as in any other form of cardiac disease when the pulmonary compensation is broken. Anginal attacks and coronary sclerosis may usually be regarded as a feature of the sclerotic form of aortic insufficiency rather than a complication. On the other hand, one of the cases under the writer's care was very subject to severe attacks of definitely anginal character, and yet at autopsy the coronary arteries were found to be clear. It is possible that in such cases the pain may be due to either vasomotor ischæmia or ischæmia due to the low mean pressure in the coronary arteries.

TREATMENT AND PROGNOSIS.

For purposes of prognosis and treatment the course of the disease may be divided into three stages.

1. Freedom from symptoms, the left ventricle performing its work perfectly without either dilatation or pulmonary stasis. In this stage the high pulse-pressure, low diastolic pressure, and collapsing pulse and throbbing arteries are, nevertheless, prominent. The only indication is to aid nature by avoidance of overstrain, overeating, alcohol, coffee, tobacco, unhygenic surroundings, and exposure to infectious diseases. When hypertrophy is good, the lesion may persist for years without producing the slightest symptoms.

Three years ago the writer examined a medical student twenty-five years old who has been in perfect health since an attack of rheumatic endocarditis at the age of ten. In spite of his lesion he has become an athlete, was a member of his class crew at Yale, and excelled in long-distance running, he smokes and takes alcohol in moderation. Cases like this may persist for thirty-five years or more (Osler), but manifest themselves sooner or later after infectious diseases or with the onset of the arteriosclerotic period of life.

In cases with arteriosclerosis, potassium iodide, 0.3 Gm. (gr. v), or sodium nitrite, 0.2 Gm. (gr. iii), three times a day, is advisable, to help check the progress of the arteriosclerosis and to keep down the blood-pressure. Occasional Nauheim baths, warm salt baths, or even ordinary warm baths are useful in promoting the vasodilation Cardiac tonicity must be maintained at all costs and dilatation must be prevented.

2. The second stage is that of dilatation of the left ventricle. This is the stage when symptoms appear, some arising in the sensory nerves of the ventricle and manifesting themselves in the forms described above—palpitation, referred pain, psychic disturbances, anginal attacks, some arising in the pulmonary circulation as a result of stasis, presenting the various forms of respiratory disturbance. According to Head, the stage of cardiac sensation never merges into the stage of respiratory distress, but disappears when the functional mitral insufficiency and the latter symptoms set in. There is a "safety-valve" action of the mitral valve.

As has been seen, the important factors producing distention of the ventricle are diminution of tone and high peripheral resistance. Treatment should therefore be directed toward counteracting these conditions. The usual cardiac procedures, rest, light diet, free purgation, should be resorted to, and, when improvement warrants, the Nauheim baths and gentle exercises free from much resistance.

Digitalis and the Nitrites.—The use of digitalis has been much disputed. Corrigan stated that "in every case of this disease in which digitalis has been administered it has invariably aggravated the patient's sufferings." Broadbent believes that it should be used with caution and that it may even precipitate sudden death, but that it is certainly indicated after mitral insufficiency has set in. Romberg thinks it should always be used with caution. It is probable that any deleterious property which the drug may possess lies in its vasoconstrictor action, and hence from *a priori* reasons it should be combined with nitroglycerin or other nitrites, or preferably strophanthus substituted. Indeed, strophanthus seems to be the drug *par excellence* in these conditions, but with this drug it is well to give nitroglycerin. The pharmacological researches of Cameron demonstrate that the nitrites possess the two properties most needed in aortic insufficiency, that of increasing tone and of dilating the peripheral vessels, and the writer's clinical experience bears out the view that, either alone or with strophanthus or digitalis, they furnish great relief and are to be strongly recommended.

A beautiful series of animal experiments recently carried out by Cloetta merits consideration.

Cloetta found that the administration of digitalis over prolonged periods caused no changes in size and strength of normal rabbits' hearts. If aortic insufficiency were produced and the animals left untreated for a year, their hearts hypertrophied and gained

80 per cent of their original heart weight, but the animals lost in strength and endurance. If they were treated with digitalis immediately after producing the lesion and the treatment continued throughout the year the hearts were smaller (hypertrophy 30 per cent.), but the hearts were almost as strong as those of normal rabbits. The aortas of untreated animals had widened much more than those of the treated.

Cloetta claims to have had equally good results in patients by early and continuous treatment with digitalis, but the matter must be studied upon a larger series of cases before attaining general acceptance. It is chiefly applicable to early rheumatic cases, though it seems probable that long-continued administration of very small doses of digitalis (0.3 c.c., or 5 minims, of the tincture) may exert this beneficial effect without producing the harmful effects sometimes met with.

For the extreme palpitation and anginal attacks, little can be done beyond the administration of amyl nitrite in the latter. Ice-bags to the precordium are often of value, as is the Finsen light treatment, galvanism of the vagus (J. O. Hirschfelder), etc. Stewart has found excellent results after a lumbar puncture, even when the cerebrospinal pressure was low. It is not unlikely that acupuncture over the neural segment afflicted might have the same effect.

3. The *third stage* is the stage of failure of the right ventricle, presenting the usual signs and symptoms except that anginal attacks, spasms of cardiac asthma, and Cheyne-Stokes breathing are a little more common than in other diseases. In the treatment of this condition the aortic insufficiency is more or less disregarded, digitalis, purgatives, and diuretics being given quite freely. The administration of nitrites is, however, still to be advised.

What can be accomplished occasionally in such cases is shown by the case cited on page 154, a farmer aged 53 years, who came under the writer's care in November, 1903, entering the hospital after a year's suffering with orthopnœa so great that he had been compelled to sleep in a chair for six months, and œdema and ulceration of the legs, as shown in Fig. 123. Under digitalis and free purgation improvement set in rapidly, and in ten weeks he left the hospital free from œdema and almost free from dyspnœa. He has remained quite well, and has continued his work as a farmer during the past five years.

On the other hand, failure of compensation is usually a more serious event than in mitral insufficiency, since the factors producing weakening of the ventricles from over-distention are more intense and more persistent. The writer has found in several instances that broken compensation in aortic insufficiency is associated with a high diastolic pressure which falls as the case improves in many cases shortly before death. It is probable that this is due to asphyxial vasoconstriction and furnishes another example of the vicious circle:

{Broken compensation / Slowed circulation / Medullary asphyxia} → {Vasoconstriction / Increased peripheral resistance} → {Increased regurgitation / Weakening of ventricle} → {Broken compensation / Slowed circulation / Medullary asphyxia}

Venesection is not indicated except when there are a considerable grade of venous stasis, high venous pressure, and dilatation of the right auricle, but in the writer's experience its results are then excellent.

In the anginal attacks and the spells of dyspnœa or for insomnia, codein, .03 Gm. (gr. ½), or morphine, .0075 Gm. to .03 Gm. (gr. ⅛ to gr. ½), hypodermically, may be necessary, but should always be used as sparingly as possible, since the habit is readily formed and the patient injures himself by feigning dyspnœa in order to get the drug.

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V.

AORTIC STENOSIS.

PATHOLOGICAL ANATOMY.

In a certain percentage of cases (10 per cent.) in which the aortic valves are diseased, the cusps become fused into a ring by which the orifice into the aorta is narrowed (aortic stenosis). Owing to the force within the ventricle, this ring is usually pushed upward into the lumen of the aorta until the orifice has a sort of dome-shaped appearance (Figs. 216 and 217, A).

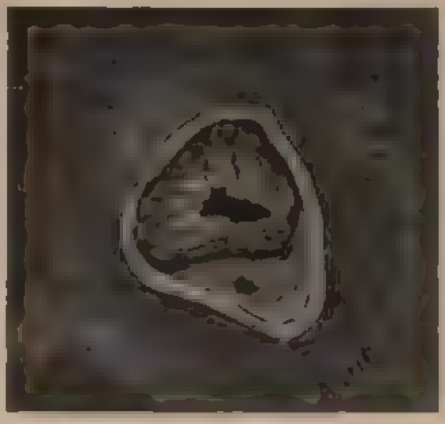

Fig. 216.—Specimen showing aortic stenosis. Viewed from above.

The inflammatory or atheromatous changes most commonly begin in the cusps separately, and the process extends until their edges become fused with an organization or atheroma at the line of union. Occasionally there is a progressive uniform diffuse sclerosis like that which often occurs in mitral lesions. The condition almost always arises from the same conditions as aortic insufficiency, but in rare cases may also be of congenital origin.

Naturally many of the manifestations depend upon the degree of stenosis, which is sometimes so extreme that a quill can barely be passed

Fig. 217.—Forms of stenotic aortic orifices. A, Lateral view of the specimen shown in Fig. 216. B, C, Aortic stenosis with edges of cusps flexible, fixed at ring (shown by broken line) but capable of movement indicated by arrows. D, E, Aortic stenosis with rigid cusps.

through the orifice. On the other hand, the orifice may be, relatively speaking, wide, and the valves retain sufficient flexibility to close it during diastole, so that a pure aortic stenosis occurs without any insufficiency

whatever (a condition present in about 60 per cent. of the cases). In the other 40 per cent. the orifice is not only narrowed but the cusps are so fused and rigid that they do not close the aortic orifice during diastole, and an aortic insufficiency is present along with the stenosis (double aortic lesion).

OCCURRENCE AND ETIOLOGY.

Aortic stenosis is by far the rarest of left-sided valvular lesions, occurring in only 5 per cent. of the 1781 Johns Hopkins cases and in 2.73 per cent. of Romberg's cases. This is in accordance with the experience of most writers. Gillespie's statistics, in which it was supposed to occur in 18 per cent. of all the heart cases in the Edinburgh Royal Infirmary, are unique and arouse the suspicion that the fault lay in the diagnosis.

The etiological factors are practically the same as in aortic insufficiency. Syphilis and arteriosclerosis play a relatively important rôle. Congenital stenosis also occurs occasionally. In rare cases there is a double stenosis,—one at the aortic orifice, and one occurring within the ventricle by the formation of a fibrous ring from the septum to the anterior cusp of the mitral valve. The disease is rare among women.

PATHOLOGICAL PHYSIOLOGY.

The changes in the circulation due to stenosis of the aortic orifice were very completely shown by Luderitz under the guidance of Prof. Gad. Luderitz found that if the aortic orifice were narrowed by the tightening of a clamp, the aortic blood-pressure might or might not fall, but the form of the

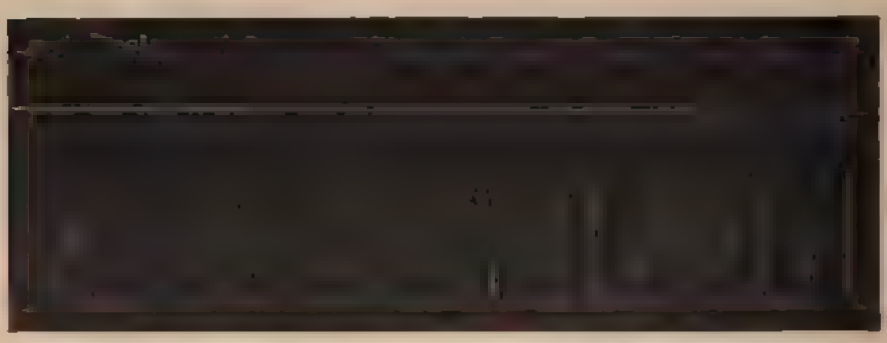

FIG. 218.—Carotid pulse and intraventricular pressure in experimental aortic stenosis. After Luderitz, *Ztschr. f. klin. Med.* xx. [illegible] marks the point at which aortic stenosis was produced. The carotid pulse (*CAR*) shows the gradual development of the pulsus tardus, with a fall in blood pressure, while the intraventricular pressure (*INT*) increases tremendously.

pulse-curve changed. The upstroke changed from sudden to gradual and slanting, ending with a broad rounded top whose summit was reached near the end of systole (pulsus tardus). This form of pulse, as will be seen, is perfectly typical of aortic stenosis, and furnishes the basis for the diagnosis.

Rise of Intraventricular Pressure.—The pressure within the ventricle, on the other hand, rises greatly, often as much at 100 per cent., without affecting the aortic pressure; for the greater part of the contraction is unable to force much blood into the aorta. The excess of intraventricular over aortic pressure is therefore much greater than in any other condition. The conditions under which the contraction takes place conform more or less to those for the execution of an isometric contraction, and the curve of intraventricular pressure comes to resemble that of an isometric contraction, the summit changing from flat to the dome-shaped, as is typical for the latter (as shown by Frank and by Huerthle). That is, the pressure does not at once reach its maximum, but rises gradually, coinciding quite well with the rise of the curve in the aorta. It is the direct communication of this progressive rise of pressure to the aorta which gives rise to the pulsus tardus, as well as the fact that the volume of blood flows into the aorta more slowly than usual. The duration of systole is prolonged considerably, seven to ten per cent. in mild grades of stenosis, ten to fifty per cent. when stenosis is extreme.

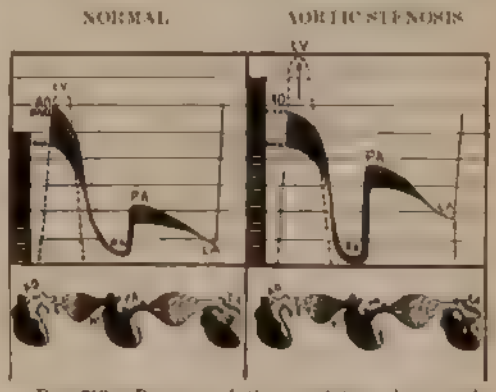

FIG. 219.—Diagram of the circulation showing the effect of aortic stenosis. The broken line indicates the intraventricular pressure. The vertical black line indicates the volume of the heart, the shaded portion representing the amount of residual blood.

When the ventricle is not able to expel its quota even by the end of systole, extrasystoles are likely to occur, and this frequently assumes the form of a continuous bigeminal pulse. Such overfilling of the left ventricle naturally leads to stasis in the auricle and pulmonary veins, with rise of pressure in these parts, pulmonary congestion, cardiac dyspnœa (v. Basch), œdema of the lungs (Welch), and secondarily also of the right ventricle. These in turn lead to dilatation and hypertrophy of the left ventricle and left auricle and hypertrophy of the right ventricle, which are usually found to be present at autopsy.

SYMPTOMS AND CLINICAL COURSE.

Aortic stenosis is probably the most chronic of all valvular lesions, and persists for years without affecting the duration of life. However, as soon as the stenosis becomes marked, so that the left ventricle has difficulty in emptying itself completely, slight exertion, excitement, or emotion brings on disagreeable symptoms, palpitation, constriction, substernal pain or anginal attacks, and shortness of breath. These sensory stimuli probably arise in the depressor nerve as the result of distention of the ventricle, for experiments of Sewall and Steiner have demonstrated that distention has this effect in animals. The symptoms at first pass off when the patient rests or leads a quiet and hygienic life, but as the disease persists they become more frequent and persistent. Sudden death is relatively common, and is probably due to acute dilatation.

Compensation. As in mitral stenosis, compensation is difficult. The left ventricle may by increasing its power continue to drive enough blood into the aorta to maintain the blood-pressure, and even to cause the pulse to resume the normal form (Fig. 223), but this is done at

an enormous waste of energy, which sooner or later brings on heart failure. Moreover, the lesion itself is slowly progressive, and this constantly increases the difficulty of maintaining the circulation. In the final stage broken compensation sets in exactly as in other advanced valvular lesions.

When aortic insufficiency coexists the circulatory difficulty is naturally increased, since the ventricle must drive an even excessive amount of blood into the aorta in order to maintain the circulation, in spite of the difficulty under which it already labors. Moreover, these are often the cases with the most advanced pathological lesions, so that the coexistence of aortic insufficiency renders the prognosis less favorable than that of pure aortic stenosis

PHYSICAL EXAMINATION.

The most striking feature upon general physical examination in aortic stenosis is the presence of a well-marked systolic thrill and bruit over the larger arteries. Over the chest there is usually a certain amount of precordial bulging The apex impulse is sometimes well marked and heaving, situated quite outside the mammillary line in the fifth or sixth interspace, frequently, however, it is not visible nor palpable. Between the apex and the sternum there is often some systolic retraction of the interspaces from the contraction of the hypertrophied right ventricle. The left ventricle hypertrophies, increasing in size along its long axis (obliquely downward).

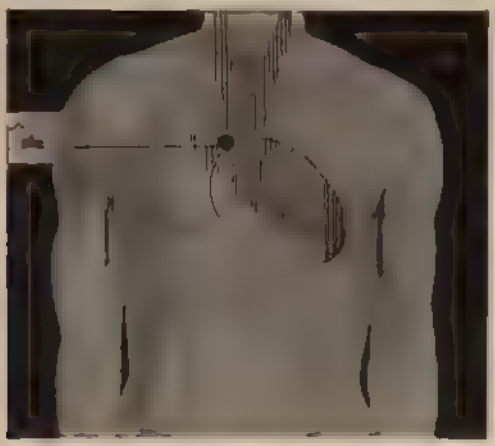

FIG. 220. Diagram showing the cardiac outline and distribution of the murmur in aortic stenosis. The parallel shading indicates the distribution of the systolic murmur and the dot indicates the point at which they are most intense

Palpation. - Palpation reveals a systolic thrill which is usually very marked and felt over the whole heart, especially over the aortic area. It is present in the carotid and brachial arteries, and is transmitted in the direction of the blood stream (see page 92). The intensity of this thrill is often the most striking feature of all the physical signs, and may far exceed that which is found in any other condition The shock of the first sound is usually felt, while that of the second is often, though not always, absent.

Percussion and X-ray examination reveal no peculiarities other than an area of cardiac dulness enlarged along its longitudinal axis, as in aortic insufficiency, but, owing to the presence of functional mitral insufficiency and dilatation of the conus arteriosus, the area of dulness may be higher and broader than in aortic insufficiency and resemble that found in organic mitral insufficiency.

Auscultation.—On auscultation one is immediately struck by the presence of a loud systolic murmur most intense over the aortic area, and transmitted thence to the first right inter-

space and along the course of the arteries, where it is, as a rule, still loud and distinct. It is also heard over the pulmonic area, body of the heart, and over the apex, but far less loudly than in the aortic area and the arteries.

This murmur is usually the loudest that is heard in any form of valvular disease, and is often heard several feet away from the patient. The mechanism of its production exemplifies perfectly the simple experiment for the production of thrills and murmurs described on page 92. Since it cannot be produced until the blood begins to flow into the aorta, this murmur does not begin until an appreciable interval after the beginning of systole (Boy, Teissier, Romberg, Weiss and Joachim) and follows the first sound in that region as well as at the apex (Fig. 221). Weiss and Joachim have recorded this murmur with their phonoscope, and find that it sets in with a crescendo character at the very end of the first sound. The crescendo continues until the crest of the carotid pulse, after which it changes to decrescendo throughout the rest of systole. The form of the carotid wave portrays the amplitude of the vibrations and the variations in loudness of the murmur. When mitral insufficiency (organic or functional) is present the mitral murmur may enter into or replace the first sound.

Fig. 221. Murmur of aortic stenosis. After Weiss and Joachim. Upper curve, carotid pulse; middle curve, heart sounds; lower curve, time [illegible] second. The second sound is practically absent. The murmur is composed of a crescendo followed by a decrescendo character loudest with the upstroke on the pulse wave.

The second sound may vary considerably in aortic insufficiency. If the valves are fused throughout their whole extent, it will be entirely absent, but if portions of the cusps remain freely movable their closure may give rise to a sound. Owing to the small excursion, this sound may not be as loud as it would be if no stenosis were present, but this factor may be more than balanced by the presence of sclerotic plaques and calcifications whose concussions may actually render the second sound louder than normal.

PULSE.

Aortic stenosis may be said to be the only disease in which the absolute diagnosis is determined by the pulse-tracing. The pulse is small, hard (high diastolic pressure), and in typical cases rises and falls very slowly (pulsus tardus). Like the curves in experimental aortic stenosis (Fig. 219), the typical radial pulse-curve (Fig. 222) shows a very oblique ascent which lasts throughout systole, the summit of the curve appearing just before the dicrotic notch. This is produced by the slow, gradual, and progressive filling of the arteries from the gradually increasing intraventricular pressure. It may be recalled that during the period of the up-stroke upon the pulse-wave blood is flowing into the aorta more rapidly than onward

to the periphery; that during the period of the plateau the inflow and outflow are equal; and during the period of fall blood is flowing onward to the periphery more rapidly than it flows into the aorta.

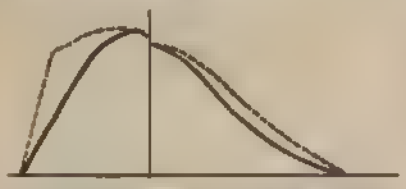

Fig. 222.—Diagram showing the pulsus tardus and the anacrotic type. Solid line, pulsus tardus showing the slow gradual rise, broken line showing the anacrotic form with sudden almost vertical rise surmounted by a plateau which takes up the greater part of systole.

The pulse of aortic stenosis, therefore, reflects the true condition, that blood is flowing into the aorta less rapidly than usual and out of it also less rapidly. However, it must be admitted that this typical form of pulse is rather rare. Most commonly, either the aortic stenosis does not reach this stage without being complicated by an insufficiency which changes the pulse form or death intervenes before these signs of inability of the heart to empty itself have set in. Indeed, many practitioners may pass through long lives of busy practice without encountering a single example of aortic insufficiency with pulsus tardus. The anacrotic pulse is so much more

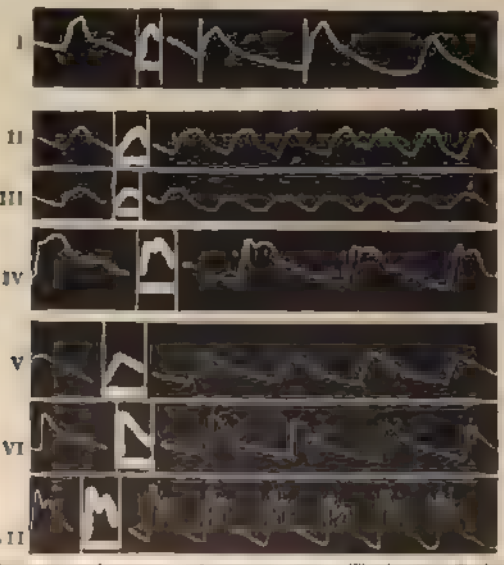

Fig. 223.—Pulse tracings from cases of aortic stenosis. The heavily shaded curve represents the systolic portion of the tracing. I. Anacrotic pulse from a case of tricuspid insufficiency but no aortic stenosis, showing quick upstroke with only the summit sloping. II, III. Pulsus tardus from a case of aortic stenosis. I. S. V. IV. Tracing from a case of aortic stenosis and insufficiency. V, VI, VII. Tracings from another case of aortic stenosis and insufficiency. V. Taken on February 28 soon after admission; symptoms of cardiac weakness well marked; maximal blood pressure 130 mm. Hg. The upstroke is gradual and sloping. VI. From same patient on March 4, after recovery from cardiac symptoms. Maximal pressure 170. The strong heart forces blood rapidly through the stenosed orifice and causes a sudden upstroke. VII. Pulse tracing taken immediately after VI, with other arm raised. The increased resistance changes the tracing to a pulsus bisferiens.

common in aortic stenosis that examples of it are given in many text-books erroneously labelled pulsus tardus. A pulse-curve with a sudden perpendicular up-stroke, however, is not a pulsus tardus, but an anacrotic pulse, whatever may be the form of its summit.

It indicates that blood is flowing into the aorta from the heart more rapidly than it is flowing out of the aorta toward the periphery, a condition which occurs in aortic stenosis only (1) when the orifice is so slightly narrowed that the hypertrophied left ventricle is able to drive blood through it with great rapidity, and (2) when the peripheral vasoconstriction is so great that, in spite of a slow inflow into the aorta, the blood still enters the latter much more rapidly than it can leave it. The former is the more common condition; and it would appear that the hypertrophy of the ventricle can usually keep pace with the advancing stenosis until a very late stage is reached. The pressure within the ventricle produced under these conditions is probably tremendous.

These facts are well illustrated in Curves V, VI, VII (Fig. 223). The first (V) was taken when the patient's heart was weak, and the blood-pressure shows a gradual up-stroke and is fairly typical of aortic stenosis. The other curves (VI and VII), taken after his heart had improved, have taken on the characters of aortic insufficiency and have lost those of aortic stenosis.

Arrhythmia.—The rhythm of the heart in man, as in animal experiments, is frequently irregular; small beats and dropped beats being frequent, due to the occurrence of a pulsus alternans or to extrasystoles arising in the left ventricle when that chamber is unable to empty itself sufficiently. Exercise, emotion, or any other form of cardiac overstrain, on the one hand, or of cardiac weakening, on the other, precipitates this irregularity.

Blood-pressure.—The blood-pressure in aortic stenosis is usually slightly elevated (maximal pressure 130 to 160 mm.), due in part to the accompanying arteriosclerosis, in part to the increase in the intraventricular pressure, especially when the heart hypertrophies.

DIAGNOSIS.

In typical cases the diagnosis of aortic stenosis is extremely simple. The presence of slow, gradual pulse, the pulse-tracings, the enlarged heart, the very intense systolic thrill, the thrill and murmur over the aortic area and arteries, and the absence or marked diminution of the aortic second sound, present a perfectly characteristic picture. In certain cases, however, and especially when there is arteriosclerosis or aortic insufficiency, it may become extremely difficult to decide whether a mild grade of stenosis is present.

CASE OF AORTIC STENOSIS.

Mrs. L. S., housewife, aged 58, entered the Johns Hopkins Hospital, April 29, 1904, complaining of heart trouble. She has always been healthy; has had no infectious diseases and never had rheumatism, but occasionally has had sore throat. She has occasionally had fainting spells and palpitation after mental excitement, and during the past year has had to void three or four times a night. Except for these symptoms she was quite well until a year before admission, when one night after a heavy meal she awoke with extreme dyspnœa, palpitation, and a feeling of extreme weakness. She had no pain, but felt considerably alarmed. Immediately after this her feet became swollen and in spite of a sojourn in bed she became subject to attacks of extreme dyspnœa. The œdema of the feet subsided, however, but reappeared after exertion.

At the time of examination the patient was propped up in bed, with slight dyspnœa. She was fairly nourished, pale, sallow, lips very cyanotic. Lungs

clear on percussion and auscultation, except at both bases, where the note is impaired and the breath sounds are accompanied by crackling rales.

Heart.—The apex impulse is barely visible in the sixth left interspace 13 cm. from the midline, from which point dulness extends upward to the third rib, as well as 3 cm. to the right of the midline. There is slight impairment of the percussion note over the sternum. A soft systolic murmur is heard at the apex and in the axilla, becoming louder however, as the sternum is approached, and maximal over the second right interspace, where it becomes rough in character. It is transmitted to the carotids but not to the subclavians. The second pulmonic sound is louder than the second aortic. There is a well-marked thrill over the base and manubrium, most marked in the second right interspace. Slight pulsation over the manubrium. No tracheal tug. The pulse is small, regular, 100 per minute. The left radial pulse is a trifle larger than the right. Tracing shows a well-marked pulsus tardus (Fig. I and II). Blood-pressure 150 mm. Hg.

The abdomen is distended but does not contain fluid. The legs are very oedematous.

Urine is reddish, specific gravity 1030, acid and contains a large number of hyaline casts.

Red blood-corpuscles 5,300,000, haemoglobin 85 per cent.; leucocytes 10,000.

During the first week she improved under rest, purgation, and digitalis, but on May 8 had a severe spell of dyspnoea not controlled by morphine or nitroglycerin, but somewhat relieved by strychnine .3 mg. (1/20 gr. hypo.). During the attack the aortic murmur was much less marked than it had been before. The cardiac outlines were unchanged. There was very slight development of fresh râles, indicative of pulmonary oedema. After the attack and the nitroglycerin there was unequal dilatation of the peripheral vessels. Cheyne-Stokes respiration developed during the night. A few purpuric areas were seen over the extremities and the sacrum.

On the next day she had another attack of dyspnoea, after which cyanosis deepened, respiration became labored, the pulse weakened, and the blood-pressure fell gradually until the patient died in the early evening.

At autopsy the three aortic cusps were found to be fused together by a calcareous cement, leaving an orifice not more than 3 mm. in diameter. The left ventricle was markedly hypertrophic, the right less so. Both were dilated. The heart weighed 600 Gm. There were slight atheroma of the aorta below the transverse arch, infarction and oedema of the lungs, left hydrothorax, left pleural adhesions, chronic passive congestion of the liver (nutmeg), spleen, and kidneys, general anasarca.

TREATMENT.

As regards treatment there is little to be said. Fortunately, the disease is very chronic in its course, especially when it begins after the period of adolescence has passed. A quiet life under the best possible hygiene conditions, with avoidance of infections, excitement, and all forms of stimulants and overstrain, usually serves to stave off the onset of symptoms for many years. When these once appear in spite of quiet, the case is practically hopeless. Absolute rest, light diet, moderate purgation, and lessening of the peripheral resistance by means of the nitrites and the Nauheim baths constitute the most important means of treatment. Digitalis is of value until the heart reaches its limit of hypertrophy, after which it merely precipitates overwork and irregularity of the heart.

In the acute attacks of acute heart failure, venesection should be resorted to promptly, in order to lessen the residual blood in the left ventricle by diminishing the inflow into it.

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VI.

PULMONARY INSUFFICIENCY.

Insufficiency of the pulmonary orifice usually occurs either as a congenital lesion or as a result of a severe endocarditis in which other valves are involved. Even as such it is a very rare disease, only 3 cases having been seen among 24,000 admitted to the medical service of the Johns Hopkins Hospital.

Lesions of the pulmonary valves had been described by Morgagni, but the first clinical cases of pulmonary insufficiency were described by Norman Chevers in 1846, and after him by Frerichs, Benedikt, Walshe, and Stokes. Barié in 1891 was able to collect detailed records of 58 cases with 24 autopsies.

PATHOLOGICAL ANATOMY.

The conditions leading to regurgitation at the pulmonary orifice may be divided into six groups:

I. Congenital malformations of the valve resulting in atrophy and deformity. The presence of only two, or, on the other hand, of four cusps does not usually bring about any leakage. In this category may also be mentioned stenosis of the orifice.

II. Endocarditic vegetations upon the valves, especially arising in very acute attacks of endocarditis with lesions of other valves.

III. Arteriosclerotic changes in the cusps, often associated with dilatation and arteriosclerosis of the pulmonary artery.

IV. Aneurisms of the cusps.

V. Ruptures of the cusps during coughing or strain, especially of cusps already diseased.

VI. Dilatation of the pulmonary artery and conus arteriosus leading to a functional insufficiency of the valves.

According to many writers, especially Gibson, a functional insufficiency of the pulmonary valve of more or less transitory duration takes place as a result of dilatation of the artery and of the right ventricle. This would naturally occur most frequently in cases of mitral stenosis with broken pulmonary compensation, and would account for the blowing diastolic murmur which is sometimes heard to the left of the sternum in these cases.

The experimental data upon this subject are more or less uncertain. G. A. Gibson has shown upon the dead heart that the pulmonary valves become insufficient under much lower pressures than are necessary to cause leaks at the aortic. He has also shown that these leaks can be prevented from occurring in the dead heart if the pulmonary orifice be prevented from dilating, as by surrounding it with a string. His studies would therefore lead one to believe that such regurgitations would occur readily in hearts whose tonicity was diminished and in which the fibres about the pulmonary orifice stretched accordingly.

On the other hand Sollman has shown in the living excised cat's heart perfused with Ringer's solution and other salt mixtures that the pulmonary orifice can withstand tremendous pressure without leaking

However, Stokes, Kolisko, Bristowe, Coupland, Litten, Chauffard, Gouget and Preble, have reported cases of relative pulmonary insufficiency, supported by autopsy. In all these cases there was dilatation of the right ventricle, and in three of them a mitral lesion with pulmonary stasis. It seems quite likely, moreover, that such a pulmonary insufficiency was present in cases W. H. (page 402) and B. I. (page 417), though the water test was not applied to the valves at autopsy.

ETIOLOGICAL FACTORS.

Barié's statistics collected from 50 cases of organic pulmonary insufficiency show that the two sexes are affected with equal frequency. It was found in patients of all ages from birth to 75 years, but 37 out of 46 cases (80 per cent.) occurred between the ages of 18 and 34 years. In 40 per cent. the disease was congenital, but in these it never occurred as the sole lesion, being usually associated with stenosis Rheumatism was the etiological factor in 16 per cent. of the cases. Puerperal infection, gonorrhœa, and the other infectious diseases rank next in frequency. There is also an arteriosclerotic group due to syphilis, alcohol, and other affections especially associated with mitral stenosis and sclerosis of the pulmonary artery.

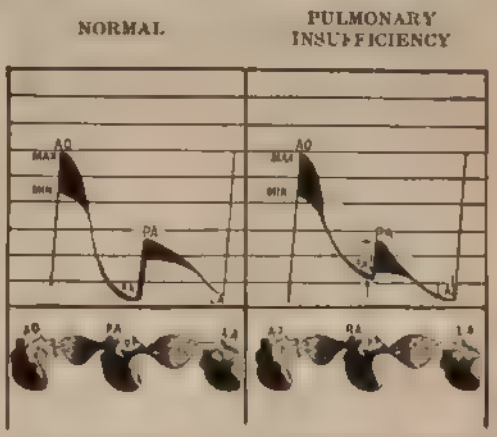

Fig. 224.—Diagram of the circulation in pulmonary insufficiency. I. Normal. II. Moderate grade of pulmonic insufficiency.

PATHOLOGICAL PHYSIOLOGY.

Pulmonary insufficiency bears the same relation to the lesser circulation that aortic insufficiency bears to the systemic circulation. The effect of the leak is to bring about a lowered diastolic pressure and an increased pulse-pressure in the pulmonary artery, accompanied by a somewhat greater systolic output from the right ventricle to compensate for the leak. The increased intraventricular pressure in the right ventricle during diastole gives rise to hypertrophy when the strain is compensated, and dilatation when the strain becomes too great. As a result of this dilatation, functional insufficiency of the tricuspid valve very readily sets in.

The results of these secondary changes are, therefore

1. To slow the circulation through the lungs
2. To cause a marked rise of pressure and stasis in the systemic veins.
3. When this occurs less blood enters the left ventricle than before. This would naturally lead to a fall in blood-pressure, but, just as in mitral stenosis it is compensated

by constriction of the peripheral vessels and the blood-pressure maintained. The vaso-constriction however manifests itself in the smallness of the arteries and of the pulse, which thus presents a striking contrast to the pulse of aortic insufficiency. The pulse-pressure also is never increased as is the rule in the latter condition.

SYMPTOMS.

The symptoms and complications are chiefly respiratory in origin: dyspnœa, especially in intense paroxysms which are brought about by slight exertion; cough and bronchitis, resulting from the poor circulation through the lungs. The intense pulsation of the pulmonary vessels weakens their walls and predisposes to haemoptysis and the expectoration of blood-tinged sputum. Phthisis is a common complication.

Fig. 225.—Distribution of the murmur in pulmonary insufficiency. The parallel shading indicates the area over which the murmur is heard. The dot indicates the point at which it is loudest; the diagram at the right illustrates its position in the cardiac cycle. The diagrams over the shaded area represent the pulsations of the systolic impulse over the pulmonary area and the systolic retraction over the right ventricle.

Palpitation is sometimes noted. Anginoid attacks and pressure at the base of the sternum are frequently met with; also pain, which, in contrast to that arising in aortic insufficiency, is more commonly referred to the right shoulder and down the right arm.

Sudden death is relatively common, sometimes resulting from over-distention of the right ventricle, sometimes from embolism in the pulmonary artery.

PHYSICAL SIGNS.

Cyanosis, as a result of the slowed circulation in the lungs, is one of the earliest signs. It is usually very marked and is liable to occur in paroxysms. Signs of bronchitis or often of bronchopneumonia are found in the chest.

Examination of the heart shows, as a rule, some precordial bulging, with well-marked pulsation of the conus arteriosus in the second left interspace, and a systolic retraction in the third, fourth, and fifth left interspaces and epigastrium, due to the vigorous beating of the right ventricle. The area of cardiac dulness is increased to the right in the transverse diameter, owing to dilatation of the right auricle. Very often it extends upwards in the second left interspace as well (dilated conus arteriosus), where it extends 5–6 cm. to the left of the midline. The area of cardiac flatness is increased to both left and right, and forms a scalene triangle extending to the right border of the sternum. On palpation the vigorous beating of the conus arteriosus may be felt in the second left interspace, also a diastolic or systolic and diastolic thrill in this region and over the right ventricle. On auscultation the sounds at the apex may be clear. The characteristic feature is the presence of a very superficial dias-

tolic murmur maximal over the pulmonary area, varying from short and soft to loud, rough and hissing in character and not infrequently musical. It is also heard along the left sternal margin, but less distinctly over the aorta. The difference is accentuated on coughing. Owing to the presence of other lesions in the pulmonary artery, there may also be a loud systolic murmur at the base, while over the base of the sternum a systolic murmur, due to the secondary tricuspid insufficiency, may also be present.

The pulse, in contrast to aortic insufficiency, with which this condition may be confounded, is small and weak; the blood-pressure is probably but little affected. Marked systolic pulsation of the veins and liver (positive venous pulse) is frequently present, due to the secondary tricuspid insufficiency. In the extremities œdema sets in readily, and there is often clubbing of the fingers and toes even in cases which are not congenital.

The following notes are taken from the records of the medical service of the Johns Hopkins Hospital:

Case of Pulmonary Insufficiency.

R. R., a colored laborer, aged 48, was admitted on Feb. 8, 1900, complaining of pain in the stomach and chest.

He had measles and whooping-cough as a child, several attacks of tertian malaria, syphilis in 1897, rheumatism in 1899, and several attacks of gonorrhœa. He uses alcohol and tobacco in moderation.

Present illness began two years ago, coincident with the onset of urethral discharge and an attack of rheumatism (gonorrhœal?). This caused him to stop work. Since then the rheumatism has become better, but he has been troubled with shortness of breath and palpitation, though these are not very severe.

Note by Dr. Henry Harris states that the patient is a well-nourished man, not dyspnœic nor cyanotic. Lungs clear except for a few moist râles over the upper fronts.

The note on the heart by Dr. Osler on Feb. 10, 1900, is as follows: "Chief impulse is in the fourth left interspace just at the nipple, also a little impulse above. The impulse in the second left interspace extends 5–6 cm. outside of the left sternal border. No impulse in the aortic area; no dilated veins; no visible pulsation of the arteries. On palpation there is no thrill There is not a very large area of cardiac dulness. The pulse is easily compressed and not collapsing. In the fifth interspace, at the apex, and over the aortic area the sounds are practically normal. In the fourth left interspace and at the nipple itself both sounds are loud. There is a short, distant, slightly rumbling murmur before the first sound, becoming distinct on moving towards the sternum.

"At the third interspace 5 cm. from the left sternal border a short, loud diastolic murmur is heard, much louder as the left sternal border is approached, maximal at the left sternal border. There is also a roughness of the first sound. The diastolic murmur disappears in the sternum, being very circumscribed. At the second left interspace 5 cm. from the left sternal border the diastolic is louder. At the left sternal border it has a maximal intensity. There is a short systolic, and a loud somewhat booming diastolic, with a rough somewhat vibratory quality. In the first interspace the murmur diminishes, being just feebly heard. In the second interspace the murmur practically abolishes the second sound, which is clearly heard at the aortic area.

"No thrill after walking about. No evidence of congenital heart disease.

"The condition is most likely pulmonary insufficiency. There is a possibility of aneurism, but firm pressure with the stethoscope far out in the second left interspace gives no sense of lifting and no diastolic shock. There is no tracheal tugging and no diastolic shock."

DIAGNOSIS.

The diagnosis of pulmonary insufficiency is rarely made during life. The history of very severe endocarditis or evidence of affection of several valves or of a lesion dating from birth leads to the suspicion of right-sided valvular disease. It is always difficult to exclude aortic insufficiency or the presence of the two lesions at once. The small size of the pulse, the absence of visible pulsation of the large arteries, the small pulse-pressure, the marked pulsation of the conus arteriosus (both against the chest wall and as shown by the fluoroscope), the retraction of the interspaces over the right ventricle, the increase in the horizontal diameter of dulness to the right and not to the left, and especially the dulness in the second left interspace furnish the basis for the diagnosis. This is also confirmed when there is pain down the right arm instead of the left. On the other hand, the congenital heart lesions—open ductus Botalli, open septum auriculorum or ventriculorum, etc.—are very difficult to exclude, and will be dealt with in connection with congenital heart diseases.

The diagnosis of functional pulmonary insufficiency is based upon the presence of a transitory diastolic murmur along the left sternal border during periods of pulmonary stasis, in the absence of other signs of aortic insufficiency. No doubt this diagnosis may sometimes be made correctly especially in cases of mitral stenosis, but it is one of which even Gibson cannot feel certain in any individual case.

TREATMENT.

Treatment is the usual procedure for cardiac overstrain of any sort,—rest, light diet, purgation, and digitalis. Venesection, by relieving the distention of the right auricle and ventricle, is particularly useful, and, as stated by Alexander Morison, yields remarkably good results in this condition.

The main hope, however, lies in bringing about the hypertrophy of the right ventricle and in preserving the balance between the strength of the right ventricle and the strain put upon it. Symptomatic treatment of the bronchitis and pulmonary complications may do much to relieve the patient.

The Prognosis is bad when pulmonary stenosis is present, but in the presence of a pure insufficiency depends greatly upon the condition of the right ventricles and the amount of cardiac embarrassment caused by the lesion. As seen from Barié's cases, patients may reach the age of seventy-five in spite of the lesion. These cases are, however, rare.

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VII.

TRICUSPID INSUFFICIENCY.

ORGANIC AND FUNCTIONAL TRICUSPID INSUFFICIENCY.

Insufficiency of the tricuspid valve occupies a unique position among the valvular lesions. In the functional form, due to dilatation of the right ventricle, it is extremely common, and indeed probably occurs at some stage in every dying or failing heart. In the organic form, on the other hand, it is rare, occurring only 16 times in 1781 cases of valvular disease at the Johns Hopkins Hospital (0.85 per cent.) and in less than 0.7 per cent. of Gillespie's cases at Edinburgh.

The organic forms occur more frequently in severe or malignant endocarditis, as is indicated by the fact that in none of the Johns Hopkins cases was it the only valve affected, mitral stenosis being present in 10, aortic insufficiency in 7 of the cases. Three valves, the aortic, mitral, and tricuspid, were involved in 7 of these cases, the pulmonary orifice once. Although severe rheumatic fever is perhaps the most frequent cause, streptococcus and gonococcus infections are relatively common etiological factors (see Chapter I), more so than in the milder valvular affections. Occasionally it occurs as a congenital lesion, the result of endocarditis during fetal life.

Anatomically the lesions of the tricuspid valve exactly resemble those of the mitral, with which they are so frequently associated, being due to vegetations, thickenings, ulcerations, hemorrhages, and occasionally tumors or malformations upon the valves.

Functional Tricuspid Insufficiency.—Our knowledge of functional tricuspid insufficiency dates from the remarkable anatomical and physiological studies of T. W. King in 1837.

King stated that "the right ventricle is liable to dilatation and that the dilatation deranges its valves.

"The last proposition is thus explained. The cavity is formed by the solid septum of the heart for its inner wall, and by a thinner, more extensive and yielding layer of muscle for its outer or right wall, whilst each of these walls affords points of attachment to the cords of the valves . . . In the progress of post-mortem examinations, I have found in hearts thus dilated, or only greatly distended by the final congestion that upon injecting the ventricle by the pulmonary artery the tricuspid curtains when stretched out were under all circumstances a great deal too small to close the opening, and it appears from careful examination that the united areas of these valvular portions are scarcely more than equal to the mean extent of the oval opening . . . I have shown that upon injecting fluids into the ventricles by their respective arteries (the semilunar valves destroyed) the left or bicuspid valve (human heart) was always seen to close completely and firmly, the curtains being so extensive as to fold together in the form of a cone or wedge within the ventricle, whilst the tricuspid valve was constantly found in its ordinary state incapable of preventing a considerable reflux. With every attempt to induce an accurate closure of this valve, its scanty and divided curtains united imperfectly or scarcely met and were only sufficient at the best to form a plane

equal to the area of the opening . . . No position in or out of water, no degree of gentleness or force, no state in anywise natural to the organ that I was able to induce, would prevent a considerable riband-like stream of regurgitation between the ill-apposed edges of the valve . . The only possible means of obtaining a nice, though weak, adjustment of the tricuspid curtains was to compress the ventricle and by the same means to lessen the extent of the valvular aperture . . . I have twice had an opportunity of experimenting on the human heart at the earliest period that propriety could admit of. In one of the cases (of which I have not hitherto spoken), after performing the experiment and eliciting results similar to those related, the heart was set aside with the expectation that its tonicity would gradually contract the ventricles and fleshy pillars which accordingly occurred. The first trial of this heart was made with warm water and the fluid was thrown in at first gently, and afterwards pretty forcibly but the regurgitation at this time was always considerable. Now upon repeating this experiment on the same heart when contracted after the lapse of a few hours, the tricuspid valve was still found to be much less incomplete, still in this case there was some refluent stream. In the second case, however, under precisely similar circumstances I obtained at least an almost perfect valvular action."

FIG. 226. The outline of a normal heart superposed upon that of a dilated heart showing the enlargement of the tricuspid orifice. Normal heart shown in light shading, dilated heart shown in black, the diameter of the orifices in white and black bars respectively.

King also confirmed these observations by extended experiments upon the hearts of a great variety of mammals and birds during life as well as after death. His experiments were repeated and substantiated by G. A. Gibson in 1880, who showed that merely narrowing the orifices by constricting them with a cord was sufficient to prevent the reflux. Francois-Franck in 1882 was able to demonstrate the production of tricuspid insufficiency in the living animal under conditions which led to cardiac dilatation, and to demonstrate its disappearance under digitalis. The frequency with which such functional insufficiencies occur in heart failure during life was shown by Friedreich, Mahot, Riegel, Mackenzie, Hirschfelder, and a host of other writers. In hearts which have been dilated for a long period there is a considerable stretching of the valvular orifice, as was already noted by King. This has lately been very clearly shown by Keith's figures of the hearts of Mackenzie's patients, in which the stretching was so great that the usual narrowing at the auriculoventricular opening had completely disappeared (Fig. 226).

PATHOLOGICAL PHYSIOLOGY.

As Rosenbach has shown, the production of tricuspid insufficiency has in itself little effect upon the systemic circulation. Blood-pressure in the arteries remains unchanged, and there is no characteristic change in the pulse. In the pulmonary circulation there may be a slight fall of pressure as a result of the regurgitation. On the other hand, this may be compensated by a slight increase in the systolic output of the right ventricle and no change may occur.

The principal effect of tricuspid regurgitation is exerted upon the circulation in the systemic veins. The blood thrown back into them at each systole causes the pressure to rise, so that in such cases the pressure may reach as high as 26 cm. H_2O (20 mm. Hg) (Hooker and Eyster). The stasis thus occurring also affects the peripheral circulation of the limbs and body, giving rise to œdema and ascites; stasis in the kidneys causing diminished excretion of a concentrated urine rich in albumen and casts, also stasis in the medulla oblongata where the accumulation of CO_2 causes a general reflex vasoconstriction. The secondary effect of this vasoconstriction is rise of general blood-pressure, further increase of the work of the heart, and increased heart failure—the vicious circle of asphyxia (see page 27).

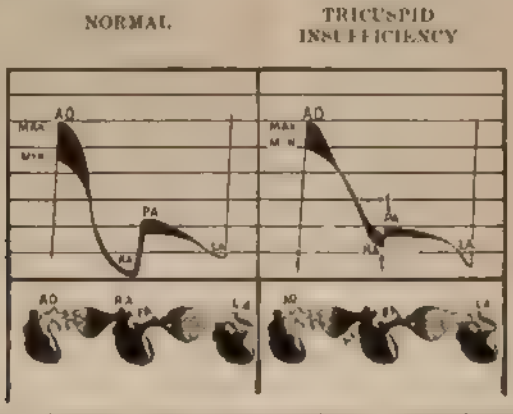

FIG. 227.—Diagram showing the changes in the circulation in tricuspid insufficiency. The arrows show the rise in pressure in the right auricle (*RA*) and vena cava, and the fall of pressure in the pulmonary artery (*PA*). The white curves represent the pulse-waves, that above *RA* showing the ventricular type of the venous pulse.

Venous Pulse in Tricuspid Insufficiency.—In contrast to the normal (negative, presystolic, diastolic, "double") venous pulse, the typical pulsation in tricuspid insufficiency is synchronous with and of the same frequency as ventricular systole (single venous pulse) (Friedreich, Riegel, Mackenzie, Hirschfelder). Since there is a free communication between auricle and ventricle, the jugular pulse-wave (Fig. 228) closely resembles the curve of intraventricular pressure, with its up-stroke and plateau during systole and its fall during diastole. In the advanced stages the wave (*a*) due to auricular systole is absent, since the auricles are paralyzed (Mackenzie).

Mackenzie states, however, that, contrary to preconceived notions, all cases with tricuspid insufficiency do not necessarily show a positive venous pulse, and in a number of his cases which at autopsy showed both organic and functional insufficiencies the positive venous pulse was absent. Mackenzie finds in these cases that the up-stroke of the wave (*v*), which is due to stagnation in the ventricle, begins earlier than usual. As the lesion increases, this wave (reflux) begins sooner and sooner after the beginning of systole, until finally it takes up the entire systolic period, and the posi-

tive or ventricular type is assumed. These observations have been confirmed in man by Gibson and Sewall, and in animals with tricuspid lesions by J. Ruhl, who found that as long as the regurgitation was slight the auricular type of venous pulse persisted, but when it became severe this gave way to the ventricular type. As Sewall states, "among patients presenting themselves for examination on account of a wide range of functional disorders, I have been struck with the uniformity with which evidences of cardiac insufficiency could be distinguished, based upon the nature of the symptoms and the character of the venous pulse . . . The *v* wave has a double crest; or rather, the wave *r*, which begins just at the moment of closure of the aortic valves, as determined by the dicrotic notch in the lower tracing, is immediately preceded by a wave which is completed during the last moments of ventricular outflow." He believes that this last-mentioned wave (the *t* wave of Bard) is produced by a slight regurgitation due to weakness of the papillary muscles, and is indicative of such regurgitation, but he does not take into account the fact that it may be present without any other signs of tricuspid insufficiency. On the other hand, as shown by Theopold, Hewlett, and others (page 75), the positive venous pulse may be present without any regurgitation at the tricuspid.

FIG. 228.—Venous pulse of patients with tricuspid insufficiency (positive venous pulse). *JUG* pulsation over the jugular vein, *BRACH* pulse in the brachial artery, *c* moment of onset of the pulse wave in the carotid artery. The tracing shows an elevation throughout systole with a very slight depression, perhaps due to fling, immediately following the upstroke. The curve corresponds almost exactly to the curve of pressure in the right ventricle.

FIG. 229.—Venous pulse of another patient. *VJD* right jugular pulse, *ACS* left carotid artery. The slow slanting upstroke indicates a slightly less smaller leak than in the preceding case. There is no fling, and hence no midsystolic depression.

SYMPTOMS.

The condition of patients with tricuspid insufficiency well illustrates the fact that this is one lesion which is not often compensated, though compensation can take place through increased suction-pump action of the right ventricle. They are usually markedly dyspnœic or orthopnœic, weak and readily exhausted by the slightest effort, often drowsy and somnolent,

Palpitation may be extreme. One of the early symptoms is pain in the region of the liver, from the stretching of the capsule. This is often accompanied by slight jaundice, and the appearance of an icteroid hue is one of the unfavorable signs in tricuspid insufficiency, since it marks an intense hepatic stasis. Gastric disturbances, loss of appetite, and indigestion are the rule and vomiting is frequent.

PHYSICAL EXAMINATION.

The patients are usually quite pale and deeply cyanotic. When secondary renal changes have set in, the face may be puffy. Emaciation and slight jaundice, the result of catarrhal cholangitis from stasis in the portal system, are among the most suggestive signs that tell the onset of tricuspid insufficiency. The veins are full and show well-marked pulsation, systolic in time and synchronous with the carotid pulse. There is often œdema of the extremities, genitalia, and back, and large ecchymoses are not uncommon. Ascites and right-sided hydrothorax are seen in the last stages of almost every case. Examination of the eye-grounds usually shows distention of the retinal veins (Black). The urine is usually scant and concentrated, and contains a large amount of albumen and casts in large numbers.

Heart.—The precordium often bulges, and the very vigorous beating of the hypertrophied right ventricle is seen in the retraction of the interspaces between the parasternal line and sternal margin. In the epigastrium and over the liver a systolic pulsation is seen and felt. Percussion shows a marked extension of the cardiac dulness to the right of the sternum, due to dilatation of the right ventricle. It often reaches 5–6 cm. from the midline, but the cardiohepatic angle remains acute. There may be or may not be extension of dulness to the left mammillary line dependent upon the presence of weakness of the left ventricle. The characteristic modification of the heart sounds is the presence of a systolic murmur which is over and near the lower third of the sternum, but may also be heard over the greater part of the heart, over the ensiform cartilage, and in the epigastrium. It is loudest in the fourth and fifth right interspaces, between the parasternal and the midline. Occasionally, as in Case J. D., this murmur cannot be heard when the patient is lying on his back or even standing, but can be elicited by causing him to bend forward to an angle of 15°. This does not increase the accidental murmur which is often heard over the entire right ventricle, nor does it augment cardiopulmonary murmurs over this area.

The murmur is often accompanied by a systolic thrill over the lower sternum and neighboring portions of the chest wall. The distribution to the right of and behind the sternum corresponds to the wall of the right auricle, the chamber into which the regurgitant stream is conducted (see Figs. 230 and 231). The area to the left of the sternum over which the murmur is loudly heard corresponds to the wall of the right ventricle. As in mitral insufficiency, it is difficult to explain the loud transmission of this murmur in a direction opposite to that of the leakage, but it seems possible that the vibrations of the valve may be communicated to the ventricular wall along the tense chordæ tendineæ. The murmur is rarely transmitted

as far as the pulmonary area, though a systolic murmur of different origin (accidental murmur) is often heard in the latter area in cases with tricuspid insufficiency as well as in others. The tricuspid murmur is, as a rule, not transmitted to the apex. Most frequently in dilated hearts there is also a functional mitral insufficiency coexisting, and it is this which gives rise to a systolic murmur at the apex and in the axilla, but this is usually less superficial than the tricuspid murmur and it can usually be differentiated from the latter. Moreover, there is, between the two areas at which each murmur has its maximum, a zone, corresponding to the interventricular septum, at which both murmurs diminish in intensity.

FIG. 230.—Distribution of the murmur and cardiac outline in tricuspid insufficiency. The shaded area indicates the region over which the systolic murmur is heard. The diagram at the left indicates its relation to the cardiac cycle. The heart is seen to be enlarged to the right. The systolic pulsation of the liver is indicated by the small diagram and the arrows.

As Hering and others have shown, a systolic murmur is not heard in all cases of tricuspid insufficiency, especially in those in which the heart is too weak to give rise to a loud sound or in which the aperture of leakage is too loud to produce one (large leaks). Sometimes the murmur has a musical character. Occasionally, as in Case W. H., in which the presence of tricuspid insufficiency was demonstrated conclusively by venous and liver tracings during life and by autopsy, peculiar diastolic murmurs are heard over the right ventricle, especially along the left sternal margin. They are sometimes blowing and sometimes rumbling (mid-diastolic in character), and may perhaps be caused by functional insufficiency of the pulmonic valves due to the dilatation of the right ventricle.

FIG. 231.—Cross section of the body, showing the paths of propagation of the murmur of tricuspid insufficiency.

Organic murmurs are frequently rough, while those due to functional insufficiency are usually soft and blowing, and sometimes barely audible. Hering states, as the result of prolonged experimental investigation, that functional insufficiency which gives rise to distinct murmurs is usually of slight grade, but when the orifice is much dilated and the leak is a large one no murmur is heard. This aphony of the valves corresponds to the condition described on page 110.

Except for the accompanying murmur which often replaces the first sound, the cardiac sounds are not greatly modified. The sounds at the

base are very considerably dependent upon the pulmonary and aortic pressures and on the degree of arteriosclerosis, and hence their relative loudness varies considerably.

Pulse.—The radial pulse in tricuspid insufficiency is usually small and weak and often irregular. The arrhythmia usually assumes the character of permanent absolute irregularity (pulsus irregularis perpetuus) (see page 75) and is accompanied by paralysis of the auricles.

Blood-pressure.—The blood-pressure is usually normal or a little below normal; but there are no characteristic features, and secondary rises of blood-pressure from medullary asphyxia are common.

The liver is usually enlarged and may extend far below the costal margin or even below the umbilicus. It is usually hard and its edge smooth, and often shows a distinct systolic pulsation (Fig. 232).

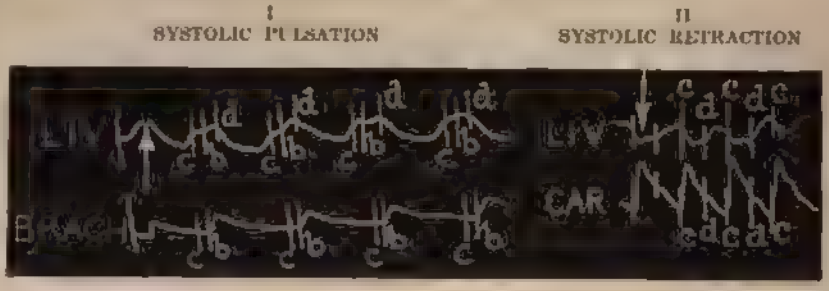

FIG. 232.—Tracings of liver pulsation. I Systolic pulsation of the liver in tricuspid insufficiency. *LIV*, tracing from the liver. *BRACH* tracing from the brachial artery. b pulse-wave in the brachial artery, c and d have their usual significance. The upstroke of the arrow indicates a protrusion, the downstroke a retraction. II Systolic retraction over the liver from a case of marked hypertrophy of the right heart. *CAR*, tracing from the carotid artery.

Ascites and oedema of varying grades may be but are not always present, dependent upon the patient's condition. "Broken compensation" does not always indicate "tricuspid insufficiency," nor *vice versa*.

CASES OF TRICUSPID INSUFFICIENCY

Myocarditis with Tricuspid Insufficiency and probably also Pulmonary Insufficiency.

W. H., colored driver, aged 48, first admitted to the Johns Hopkins Hospital on May 12, 1896, complaining of swelling of the feet and shortness of breath. He had always been healthy except for measles and chicken-pox in childhood and malaria in 1861. Gonorrhœa at 33 but no lues. Drinks and smokes in moderation.

Present illness began during the past winter, with gradually developing shortness of breath, especially on exertion. After such attacks the extremities would swell very much. A few days before admission his testicle also began to swell.

On examination by Dr. Thayer at this time he was found to be a well-formed colored man, mucous membranes of good color. Lungs clear except for moist râles over the right front. The apex was thin in the sixth interspace at the mammillary line. The first sound was feeble but no murmurs were heard. The abdomen was full; liver and spleen not palpable. Slight oedema of the extremities. The oedema disappeared under rest and digitalis. The patient gained in strength and was discharged in three weeks. He returned again three years later, with similar symptoms, and again made a rapid recovery. On this admission the liver was felt by Dr. McCrae. He was treated in the hospital repeatedly during the next few years, always presenting

about the same clinical picture. On Dec 9, 1903, the apex was 14 5 cm to the left of the midline, and Dr Thayer noted that the sounds were clear in the tricuspid area There was, however, a soft diastolic and a rumbling presystolic murmur heard over the heart between the left parasternal line and the sternal margin (pulmonary insufficiency). When he first came under the writer's care in July 1904, during a similar attack of cardiac failure, this diastolic murmur, and indeed all the other murmurs, had disappeared, the heart sounds were very feeble and the heart action irregular As his condition improved under treatment, the former murmur reappeared and increased to about the previous intensity, though heard only with the larger beats During the next admission a few months later the rumble was definitely mid-diastolic and very rough

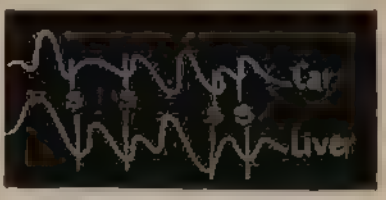

Fig 233 -Systolic pulsation of the liver of patient W H Car, carotid arterial pulse, s, onset of ventricular systole

Blood-pressure during these admissions ranged from 130 to 160 mm. Hg.

He was readmitted for the last time in October, 1905, the sounds being about as before, the oedema somewhat greater. There was severe right-sided hydrothorax Venous tracings showed a positive venous pulse of the ventricular type, and there was systolic pulsation of the liver (tricuspid insufficiency) (Fig 233). The blood-pressure during this admission was 110 mm., but rose to 130 mm on the day before death.

Autopsy showed dilatation of the right auricle and ventricle, dilatation of the pulmonary artery, marked sclerosis of the coronary arteries, very marked chronic fibrous myocarditis (cardiosclerosis), and relative tricuspid insufficiency There was marked cardiac hypertrophy, the heart weighing 620 Gm There were also chronic passive congestion of the viscera, cirrhosis of the liver, chronic interstitial nephritis, chronic fibrous pleurisy, and acute gastritis There were no valvular lesions and there was no tricuspid stenosis to account for the middiastolic rumble It is quite probable that there was during life a functional pulmonary insufficiency.

Case of Mitral and Tricuspid Insufficiency

J D., painter, aged 69, came to Johns Hopkins Dispensary complaining of swelling of the limbs He has always been healthy except for inflammatory rheumatism off and on during the last twenty years Denies venereal disease. Has not worked during the past twenty years

He has had swelling of the feet and legs after exertion during the past four years, some shortness of breath, but can always sleep without a pillow His legs and penis have been swollen for the past month

The patient is a well-nourished man, looking much younger than he acutally is. His color is a trifle sallow but not icteroid. Pupils equal. No glandular enlargement No lead line on the gums, in spite of his occupation The chest is clear on percussion and auscultation except for a few wheezing râles at the bases

The heart is markedly enlarged, dulness extending to the anterior axillary line in the fifth left interspace, above to the middle of the second left interspace and 5 cm. to the right of the midline At the apex the first sound is replaced by a blowing systolic murmur heard distinctly throughout the entire left axilla, this diminishes in intensity to the right of the mammillary line When the patient is standing and bending forward at an angle of 45° a loud blowing systolic murmur of different character is heard over the entire tricuspid area, but this is not evident in any other position In the pulmonic area there is a loud blowing mesosystolic murmur also heard in the second right interspace, but not transmitted to the carotid arteries. The heart's action is somewhat irregular, the jugular veins are distended but do not pulsate; the venous pressure, as shown by Gaertner's method, is high. (The veins of the back of the hand and wrist do not empty until the hand is about 20 cm above the level of the heart.)

The liver is not palpable. There is little if any fluid in the abdominal cavity. The scrotum and penis are markedly œdematous as are also the legs and thighs.

The patient entered the hospital, where he died of heart failure a few days later.

DIAGNOSIS.

The absolute diagnosis of tricuspid insufficiency depends upon the presence of a dilatation of the right auricle (increased dulness to the right), a systolic murmur loudest at and about the base of the sternum, a positive venous pulse of the ventricular type, and an enlarged liver with systolic pulsation.

As has been seen above, these features are not always present. Hering has summed up the whole question in the following conclusions

1 A large tricuspid insufficiency may give no murmur, but small regurgitations usually give distinct murmurs

2 A small tricuspid regurgitation may cause no change in the venous pulse, but a large leakage gives rise to a positive venous pulse of the ventricular type Hence,

I. Loud murmur + auricular (presystolic, diastolic, double physiological) venous pulse = slight tricuspid regurgitation

II. No murmur + positive ventricular venous pulse + systolic pulsation of liver = severe tricuspid regurgitation.

TREATMENT.

François-Franck showed, in his experiments upon functional tricuspid insufficiency, that the administration of digitalis caused the signs of insufficiency to disappear This is in perfect harmony with the clinical experience that "broken compensation" (and tricuspid insufficiency) is in general the signal for digitalis, and the administration of this drug furnishes the main therapeutic measure Absolute rest is necessary for prolonged periods, but after the tricuspid insufficiency has persisted for months in spite of it, it is useless to reduce the patient to a permanently bedridden condition in the hope of final recovery. It is better to render his life as pleasant as possible under the conditions, to let him sit up and move quietly about the house, go driving, or indulge in other pleasant diversions which do not entail exercise, effort, or excitement. It must not be forgotten that worry and nervousness bring on palpitation and cardiac overstrain almost as readily as does exercise; and, conversely, mental diversion and cheerfulness assist in re-establishing conditions favorable for cardiac recovery. The important feature in this phase in the management of the case is the avoidance of dyspnœa The simple methods of counting between steps on a staircase or of taking for one's gait one step for each inspiration may give the patient considerable latitude for accomplishment without strain or injury.

Diet should always be light, partly to avoid the strain on the heart, partly on account of the disordered digestion, gastritis, and catarrhal jaundice, which are entailed by portal stasis.

The bowels should be kept open with saline purgatives and several movements a day should be secured

In stages of acute heart failure when the venous pressure is high and the right auricle much distended, venesection should be resorted to promptly and continued until the right border of the heart has receded.

The best results are obtained when venesection is accompanied by intravenous injection of strophanthin (½ mg.) (see page 177) and this followed by free purgation and digitalis.

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VIII.

TRICUSPID STENOSIS.

OCCURRENCE AND ETIOLOGY.

Stenosis of the tricuspid orifice belongs to the rarer valvular lesions, and also to the group which rarely occurs alone. In the 24,000 cases which have been admitted to the Medical Service of the Johns Hopkins Hospital tricuspid stenosis has been found in only seven cases, in all of which other lesions were present. W. W. Herrick has recently given the following statistics from 187 cases collected from the literature:

SUMMARY OF REPORTED CASES.

Sex.	
Male	38
Female	133
Sex not known	16
Age.	
10 to 20 years	16
20 to 30 years	59
30 to 40 years	38
40 to 50 years	28
50 to 60 years	10
60 to 70 years	6
Not known	30
	187
Previous History.	
Rheumatism	61
Doubtful rheumatism or chorea	11
No rheumatism	33
Not known	82
Association of Valvular Lesions.	
Tricuspid alone	14
Tricuspid and mitral	102
Tricuspid and aortic	64
Tricuspid and aortic and pulmonary	1
Tricuspid and endocardium of left auricle	1
Tricuspid, mitral, and pulmonary	2
Total cases	184
Cases showing adherent pericardium	12

In Leudet's series rheumatism was an etiological factor in over 50 per cent., puerperal fever in 5 per cent. Syphilis has also been assigned as a causal factor.

In the cases in which the tricuspid stenosis follows the mitral stenosis the same etiological factors are concerned as for the single lesion. In view of the work of Goodhart, Roy and Adami, and Weber and Deguy quoted above (page 359), it is not unlikely that the overstrain of the right ventricle, brought about by the latter conditions, leads to œdema and hemorrhage into the tricuspid valve, and that these processes usher in the fibrosis. In other words, the mitral stenosis itself becomes an etiological factor in the tricuspid lesion, and the pathological process completed in the mitral is now transferred back one step in the circulation and repeats itself in the tricuspid.

Occasionally, as in a case reported by Gairdner, a fibrinous ball, a tumor, or a hemorrhage into the valve may assist in producing the stenosis.

A certain percentage of the cases are congenital in origin.

PATHOLOGICAL ANATOMY.

The anatomical changes in the valve are exactly similar to those which occur upon the mitral in stenosis of that orifice: a progressive fibrosis accompanied by fusion of the cusps along their line of closure, and gradual web-like extension of the valvular membrane, which grows downward between the shrunken chordæ tendineæ forming an elongated funnel with narrow outlet.

The liver is usually enlarged, though in some cases it may be smaller than usual, owing to the cirrhotic changes and perihepatitis which result from the prolonged stasis.

PATHOLOGICAL PHYSIOLOGY.

The changes which tricuspid stenosis produces are exactly similar to those already seen in mitral stenosis, except that they affect the systemic veins instead of the pulmonic. The filling of the right ventricle is retarded. The amount of blood which enters it passively in early diastole is diminished, and the amount driven in by the auricle is increased. The auricle thus begins to hypertrophy.[1] Its strength increases, and the presystolic wave which it produces in the venous pulse increases in size. In well-marked cases the force of auricular contraction may be great enough to produce a definite presystolic pulsation in the liver with a wave exactly similar to that found in the vein (Mackenzie).

When the tricuspid orifice is narrowed to such an extent that the increased force of the auricle no longer empties the latter, the auricular contraction begins to drive the blood back into the veins and to increase the already high venous pressure, thus still further impeding the circulation through the heart and lungs, so that the aeration of the blood is greatly interfered with and marked cyanosis produced. This in turn gradually predisposes to polycythæmia (red blood count 8,000,000 to 9,000,000). The latter condition causes increased viscosity of the blood, and still further increases the burden upon the heart. On the other hand, the

[1] The right ventricle is almost always hypertrophied in tricuspid stenosis, owing to the presence of mitral stenosis and tricuspid insufficiency.

hypertrophy of the right auricle gradually reaches its limit, and when the venous pressure becomes too high from exercise or other cause, this chamber becomes dilated and paralyzed, and the presystolic wave disappears from the jugular and liver pulse (Mackenzie). Unlike lesions of other valves, no further compensation is now possible, and only rest of the heart can prevent the over-distention of the veins. Consequently slight overstrain results at once in venous stasis, œdema, etc., which may pass off readily when the patient is at rest.

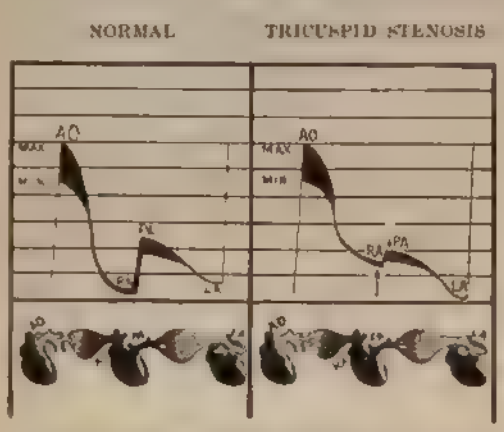

FIG. 234.—Diagram showing the changes in the circulation in tricuspid stenosis. The arrows indicate the rise in venous pressure in the right auricle *RA* and the vena cava and the fall in pressure in the pulmonary artery *PA*. The pressure in the left auricle and ventricle may remain unchanged or may fall.

The liver is often, though not always, enlarged; and a pulsation presystolic in time may be felt in it as long as the right auricle is beating strongly (Mackenzie). Œdema, ascites, and hydrothorax may be present as in other cardiac diseases.

The pulse is usually small because the peripheral arteries are constricted in order to maintain the blood-pressure, which may be perfectly normal. The rhythm may continue regular or may become irregular as the disease advances.

Still more common are attacks of heart failure and dropsy. In many cases, notably those of Shattuck and Mackenzie, such attacks may recur at intervals during a decade or more. At first the condition yields readily to rest and treatment, but later the attacks become more and more frequent and persistent.

SYMPTOMS.

Fortunately for the patients, the course of tricuspid stenosis is usually a chronic one, the development of the lesion generally lagging behind the concomitant stenosis of the mitral or the other lesions that may be present. As a consequence, the lesion may be present for a number of years without manifesting any signs other than cyanosis, and no symptoms whatever. Osler quotes a case reported by Hirtz and Lemaire who was known as "l'homme bleu" for two years before he developed any symptoms. On the other hand, in the case mentioned by Shattuck there was said to be "no cyanosis."

There is nothing pathognomonic about the symptoms. Dyspnœa on the slightest exertion sets in and becomes progressively worse. Pain down either arm is relatively common, occasionally pain about the right side and abdomen due to distention of the auricle or of the liver. Sudden death is quite common.

PHYSICAL SIGNS.

On inspection the extreme cyanosis is striking, and there may be dilatation and accentuated pulsation of the veins. When carefully timed this pulsation is seen to be presystolic, and is often a "double" pulse of the physiological type. Those characteristics are brought out more clearly by a venous tracing. In long-standing cases the fingers may be clubbed.

The lungs usually show signs of bronchitis, œdema, or often of tuberculosis. Pulmonary infarction, with the presence of areas of consolidation and the expectoration of dark red or "prune-juice" sputum, is relatively common. In the physical examination of the heart the real lesion is often overlooked. Except for the systolic retraction over the right ventricle, there may be nothing abnormal on inspection. The area of cardiac dulness is increased to the right, corresponding to the dilated right auricle, occasionally also to the left, as a result of concomitant lesions other than the tricuspid stenosis.

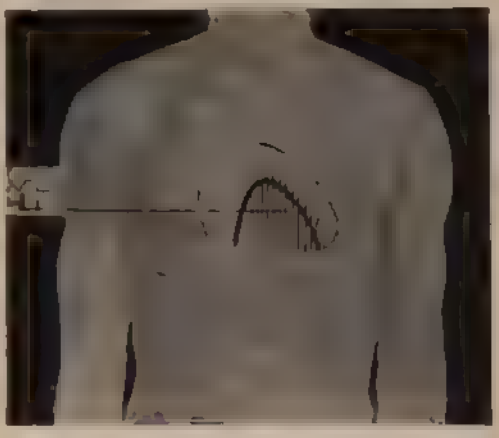

FIG. 235.—Cardiac outline and distribution of the presystolic rumble and snapping first sound in tricuspid stenosis.

Palpation sometimes reveals a presystolic thrill over the lower part of the sternum and just to the left of the latter, but it is rarely as distinct in the former situation as in the latter (due to concomitant mitral stenosis). The shock accompanying the first sound over the right ventricle may be *tapping*. The second pulmonic shock is usually less marked than might be expected to result from the lesions present.

The *characteristic sign on auscultation* is the presence of a short *presystolic rumble*, which is maximum over the base of the sternum and different in character from the presystolic rumble heard at the apex. There is also a snapping character to the first sound in this area, and it may be accompanied by a tricuspid systolic murmur. This murmur is, however, often absent, indistinct, or merges so gradually into the mitral murmur that its existence is not noted. Except when other lesions are present the second aortic and pulmonic sounds are not as loud as might be expected.

DIAGNOSIS.

So indistinct are the murmurs due to the tricuspid lesion and so completely are they overshadowed by those of the mitral or other orifices that the diagnosis before death was made in only six of Leudet's 114 cases. The correct diagnosis has been almost equally rare since then. It may be made with certainty in the presence of marked cyanosis, dilatation of the right

auricle (increase of dulness to the right), presystolic thrill and rumble, and presystolic liver pulsation. But these signs disappear as the auricle begins to weaken, and in very many cases the existence of the lesion is one that can be suspected rather than proved.

Case of Tricuspid Stenosis.

The following notes are from one of the rare cases in which the diagnosis was made during life. This diagnosis was made by Professor T. B. Futcher, who has kindly permitted the writer to make use of the notes.

Mrs A. J., aged 37, entered the private wards of the Johns Hopkins Hospital on April 30, 1909. The family history was negative. She was not a blue baby, has been healthy since childhood, but subject to occasional sore throat. She has never had acute articular rheumatism. At the age of nineteen she had an obscure fever lasting several weeks.

She has been somewhat short of breath for the past nineteen years, and since an attack of grippe about twelve years ago has complained of palpitation on exertion or after eating. These symptoms became much more marked four years ago, when oedema of the feet and ankles and cyanosis appeared for the first time. This condition passed off under treatment, but returned again two years later, again passing off, only to return with increased severity eight weeks before admission. During this attack she has been blue and has had severe orthopnœa.

Note by Dr Futcher, May 1, 1909. "Patient is of short stature, a little overstout; very marked cyanosis of ears, lips, cheeks, and finger-nails, although this is nothing as compared with the day she reached Baltimore. There is a distinct jaundiced tint to the face and sclerotics. Propped up in bed, considerable dyspnœa. Tongue moist, only a trifle coated, pupils normal size and equal, react to light and accommodation.

"Still impossible to count pulse at wrist, although very faint beats are occasionally appreciable. Thorax well formed, expansion good and equal on both sides. Lower left axillary region expands slightly less than right. Lungs. Right side clear throughout front and axilla on percussion. There is an occasional crackling râle heard at the base. Fairly numerous fine moist râles audible throughout whole back. Left lung (in semi-recumbent posture) flatness reaches to level of fourth interspace in anterior axillary line. In midaxillary line it reaches nearly to apex of axilla, and in posterior scapular line to a point about 3 cm. above left scapula. Slight movable dulness in front with change of position. On auscultation, breath sounds are harsh above and below clavicle, as in compensatory breathing. Below level of flatness there is absence of vocal fremitus and distant tubular breathing and distant nasal quality of the voice sound.

"Heart. Point of maximal impulse seen and felt in fifth interspace 11 cm. to the left of the midsternal line and just in the mammillary line. There is very slight precordial bulging, but practically no pulsation or heaving. Systolic shock distinctly tapping at apex; no definite thrill. Relative cardiac dulness commences at the upper border of the third rib, in fourth right interspace, extends 8·5 cm. from midsternal line, and merges into the fluid flatness to left, but apparently dulness extends considerably outside of midline. There is no apparent Rotch's sign to the right. The absolute cardiac dulness begins at the upper border of the fourth rib at the left sternal margin, extends to right sternal margin at level of fourth rib and to point of maximal impulse in fifth left interspace. On auscultation, the first sound is very snapping at apex. There is as yet no definite presystolic murmur, but there is a slight echoing rumble in diastole. There is no systolic bruit at the apex. The second sound is not audible here. In the fourth and fifth interspace at the left sternal border the snapping quality of the first sound is even more marked than at the apex and the tapping systolic shock is very striking here. The second sound is audible and there is definitely reduplicated. There is no rumbling presystolic murmur here. In diastole, however, there is, on very careful auscultation, a faint, soft, prolonged, blowing diastolic murmur. At the aortic area yesterday there was a faint systolic thrill. It is just perceptible this morning. The first sound is audible and is accompanied by a very rough systolic bruit transmitted upwards to sternoclavicu-

lar articulation. The second aortic is quite loud and, if anything, accentuated. There is no aortic diastolic brut heard here. Pulmonic sounds clear, second pulmonic accentuated. The external jugulars are only slightly distended.

"Liver.—Absolute flatness extends from sixth rib to a point apparently on a level with the costal margin in right mammillary line. In median line it reaches only to tip of ensiform. Owing to œdematous abdominal walls, it is not possible to palpate for liver edge. No visible or palpable liver pulsation.

"Abdomen moderately distended, walls œdematous, tympanitic in elevated and flat in dependent portions. Undoubtedly some ascites. There is very marked œdema of dependent portions of trunk, moderate of arms and hands, very marked of thighs and legs.

"Over dorsal surfaces of both wrists there are quite numerous pin-head sized petechiæ."

The urine was very scant (300 c c) of orange color, specific gravity 1015, acid, contains a trace of albumin and many hyaline and finely granular casts.

Her chest was aspirated by Dr Henry on May 1, and 500 c c of dark straw-colored clear fluid removed. She became worse, however, and her kidneys refused to act. On May 3 her pulse became irregular, cyanosis increased, and the petechial eruption on the dorsum of wrists became more extensive. She died at 3.15 P.M.

Intra vitam diagnosis by Dr. Futcher: Aortic stenosis and insufficiency, mitral stenosis, probable tricuspid stenosis.

Autopsy showed tricuspid, mitral, and aortic stenosis, dilatation and hypertrophy of the auricles, contraction and atrophy of the ventricles, chronic passive congestion of all the tissues except the lungs, generalized œdema pleural and pericardial effusion compression and atelectasis and œdema of the lungs, hemorrhagic infarctions of lungs, acute diphtheritic hemorrhagic colitis, generalized narrowing of arteries and thickening of veins.

TREATMENT.

Except for rest, purgation, and palliative treatment, little can be said in this condition. Digitalis is sometimes of value to restore tone to the auricle and increase the force of the ventricular contraction, but it very frequently fails. In the spells of acute heart failure a free venesection may ward off impending death by lowering the venous pressure, relieving the heart failure, and by diminishing the viscosity of the blood may afford more lasting relief. Free purgation is often also of great benefit, because it may lower the pressure in the veins.

PROGNOSIS.

The prognosis depends entirely upon the degree of stenosis and the rapidity of its progress. As has been said, this is frequently very chronic. Mackenzie's famous case, which is typical, was a woman whose lesion probably dated from an attack of rheumatism in 1880, at the age of twenty-nine. In 1892 she complained of weakness and shortness of breath, and at that time the liver showed a presystolic pulsation. She was subject to numerous temporary attacks of extreme heart failure and died in 1899. However, this woman was under excellent care during the last seven years of her life, and lived a tolerably discreet and hygienic existence. Had she been compelled to do heavy work her life would probably have been much shorter.

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[1] Probably intended to be, "with Report of a Case."

IX.

PREGNANCY AND LABOR IN CASES OF HEART DISEASE.

PULSE-RATE AND BLOOD-PRESSURE.

The effect of pregnancy upon the heart is influenced by several factors. The gradual pushing of the diaphragm as the uterus grows causes the heart to assume a more transverse position (raising the apex to the fourth interspace in 28 out of 35 cases observed by Stengel and Stanton), and thus placing it in a position which embarrasses its action. Moreover, a reflex vagus inhibition is often present, which causes the pulse-rate to become slowed. Blot has reported a pulse-rate as low as 36; 40 per cent. of Vegas's cases were slow, but only 26 per cent. of Skabo's cases were below 75 per minute. There is also an increase in the width of the blood channel through the uterine vessels, which is manifested by the presence of a dicrotic pulse. In order to overcome these factors and to keep up the equilibrium of the circulation, the heart is compelled to put forth increased efforts. Slemons and Goldsborough in a most careful series of observations have found the following figures, which accord well with the previous findings of O. Fellner, Stengel and Stanton, and Vogeler.

They found the following figures:[1]

	Blood-pressures.				Pulse-rate.	Cardiac output P.P. × P.R.	Work of heart. Mean pr. × pulse-[illegible]
	Max.	Min.	Pulse-pressure.	Mean pressure (min. + ⅓ P. P.)			
Normal... (Erlanger)	110	65	45	80	72	3240	259,000
Pregnancy...	127	74	53 49 in primig. 60 in multip.	91.6	80.5	3325 (primig.) 5332 (multip.)	Primig. 195,000 to 429,000 Multip. 421,000 to 1,065,000
Puerperium..	115	72.5	42.5	86.5	70.5	3000	Primig. 290,000 to 327,000 Multip. 156,000 to 337,000

HYPERTROPHY.

This prolonged increase in work was supposed by Larcher to bring about hypertrophy of the heart, a fact which has found some support in the weighings of certain observers; but the more careful work of W. Müller and of later observers (average weight of heart during pregnancy 227 Gm.)

[1] While this method of calculation is not intended to be regarded as quantitatively accurate (see p. 24), it shows the qualitative changes fairly well.

fails to substantiate this view. The increase in size supposed to represent hypertrophy is probably due in part to dilatation of the heart, and in part to the apparent increase in cardiac area which occurs when the heart lies in a more transverse position. However, a very slight hypertrophy, like that of the athlete's heart, arising from the slightly increased work of the heart during nine months, would not be surprising. During labor an additional strain is thrown on the heart, but this is of comparatively short duration.

FUNCTIONAL TRICUSPID INSUFFICIENCY AND OVERSTRAIN DURING LABOR.

James Mackenzie has shown, moreover, that the dilatation during pregnancy affects the right heart particularly, and that in very many cases even of otherwise normal women a definite insufficiency of the tricuspid valve may appear, disappear, and reappear according to the condition of the patient. The presence of this insufficiency is shown by both the positive venous pulse and the systolic murmur in the tricuspid area. The effects during the labor pains are exactly comparable to those of heavy lifting, defecation, etc. (exercises of strain of maximal intensity), and are accompanied by forced expiration with glottis closed (Valsalva's experiment) as well as by very general muscular contractions. Dr. Slemons informs the writer that during the labor pains there is often a rise of fifty millimetres of mercury in the maximal pressure, though these elevations are of short duration. It is therefore not surprising that some hearts should fail and that pulmonary œdema should be an occasional complication, especially in mitral stenosis where the pulmonary circulation is already engorged. It is perhaps surprising, however, that so few cases actually succumb during the strains of labor. Schlayer's results typify the general experience in this regard. He lost eight out of twenty-five cases (32 per cent.) of severe heart disease, but only two of these (8 per cent.) died during labor. From this, as well as from the work of Slemons and Goldsborough and the metabolism experiments of Williams and Slemons, it would appear that the act of labor itself does not impose a much more severe strain upon the organism than that arising during the course of pregnancy.

CAUSE AND FREQUENCY OF DEATH FROM LABOR.

The immediate cause of death during labor is usually pulmonary oedema from failure of the left ventricle. However, as above stated, only about one-fourth of the fatal cases die during labor, the greater number surviving some days, weeks, or months. In the cases of mitral stenosis, apoplexy or cerebral embolism is not uncommon, owing to loosening of thrombi which form in the left auricle during the periods of stasis.

As regards the results obtained by different writers in cases with heart lesions the greatest divergence is found. The following represent the mortality reported by various writers: Macdonald 61 per cent., v. Guerard 34 per cent., Lubinsky 60 per cent., v. Leyden 55 per cent., Schlayer 48 per cent., Wessner 49.3 per cent., Lwoff 12 per cent., Gusserow 6 per cent., Jess 31.5 per cent., Wiesenthal 12.5 per cent., Schneider 7.1 per cent., Muller 3 per cent.

A very careful study of these conditions has been made by O. Fellner in Schauta's clinic in Vienna. Fellner noted that the percentage of heart cases reported in obstetrical clinics was far too low for the general prevalence of cardiac disease, and upon careful routine examination found that about six cases out of seven of compensated heart diseases were actually escaping detection in the clinics. So little effect had heart disease made upon the course of pregnancy and labor! Of the cases that had been recognized in the obstetrical clinic in ten years he found the following:

	Cases.	Mother died.	Child died.
Mitral insufficiency,			
Compensated	26	0	3
Uncompensated	14	1	2
Mitral stenosis,			
Compensated			
Uncompensated	4	0	3
Mitral stenosis plus insufficiency,			
Compensated	10	0	2
Uncompensated	17	1	10
Aortic insufficiency,			
Uncompensated	2	1	1
Aortic insufficiency plus mitral insufficiency,			
Compensated	3	0	1
Uncompensated	5	0	4
Total	81	3	26

In the 900 cases occurring since his own routine examinations of the heart were instituted, he found:

	Cases.	Mother died.	Child died.
Mitral insufficiency,			
Compensated	14	0	4
Uncompensated	1	0	0
Mitral stenosis plus insufficiency,			
Compensated	3	0	1
Uncompensated	1	1	0
Aortic insufficiency plus mitral insufficiency,			
Compensated	1	0	0

FACTORS INFLUENCING PROGNOSIS.

These statistics from unselected cases are much more favorable than the previous reports would indicate, and are in accordance with the conclusions of Hicks and French that few women with heart disease are sterile, that they are not particularly liable to abort, and that most of them bear

children well. Blacker, in a most excellent *résumé* of the subject, coincides with these opinions, but finds 53 deaths (12 per cent.) in 453 cases of heart disease taken from the literature.

Most writers believe that the variety in the results is due to the severity of the cases which happen to be encountered, or at least recognized, but the excellent statistics from Schauta's clinic would indicate that skill in the management of the case plays a considerable rôle. It must not be forgotten that the recognition of an organic valvular lesion in a pregnant woman may be by no means easy, for the functional or accidental systolic murmurs at the apex, occurring during pregnancy, may closely simulate those from an organic mitral insufficiency; and, unless their disappearance is noted by the end of the puerperium, this discrepancy may not be noted. The constancy of the murmur, its roughness, its transmission to the axilla and the increase rather than decrease in intensity at times when the condition of the heart improves favor the diagnosis of an organic mitral insufficiency, while in the presence of a soft murmur occurring with a dilated heart, a rapid pulse, and a break in compensation the presumption is temporarily in favor of the more common functional insufficiency. The diagnoses of mitral stenosis and of aortic insufficiency are probably more uniformly correct and present less difficulty.

Broken Compensation in Pregnancy.—On the other hand, it may be difficult to judge when compensation should be considered broken. The pushing up of the diaphragm by the pregnant uterus causes some shortness of breath; anæmia is also a factor. The pressure upon the pelvic veins may give rise to œdema of the feet and legs and even of the genitalia. And, moreover, a relative tricuspid insufficiency of muscular origin may be present as a result of the pregnancy without organic lesion, but may nevertheless give rise to the same signs and practical effects as the latter.

The diagnosis of broken compensation in pregnancy therefore depends upon signs which are relative rather than absolute, since, as Mackenzie shows, a certain degree of broken compensation is an almost normal phenomenon in the later months of pregnancy. This again is relative, for some women are almost as active throughout pregnancy as at other times, while other quite normal women may be almost invalids throughout the entire period. It is upon degree rather than upon actual symptoms that the diagnosis of a pathologically broken compensation should be made. Dyspnœa and cyanosis on very slight exertion, such as quietly walking a distance of a few hundred yards or less, walking up a few stairs, etc., and the presence of a small rapid pulse, persistent cough, enlargement of the liver, and œdema of the feet and legs may be regarded as the most important symptoms. The earlier in pregnancy they occur the more alarming they are. Persistent dyspnœa or orthopnœa and cyanosis alone, especially in the presence of a valvular lesion, are in themselves most significant and should warrant immediate attention.

MANAGEMENT OF CASES OF HEART LESIONS IN PREGNANCY.

The correct management of a case of heart lesion complicated by pregnancy is, as stated by Blacker, to treat the heart disease without regard to the pregnancy until the break in

compensation is seen to persist, and then to terminate the pregnancy. In other words, as long as compensation is good the patient should merely be carefully watched but no medication need be resorted to. At the first signs of cardiac weakening and dilatation (dyspnœa and cyanosis, etc., on slight exertion) absolute rest should be insisted on and digitalis or strophanthus should be given. This procedure should be insisted on even if the diagnosis of organic valvular lesion is not definite, for these procedures will afford quite as much relief in cases of functional tricuspid insufficiency. Moreover, they should be repeated at the slightest indication (see page 180), especially toward the end of pregnancy. It is advisable in such cases to give a few prophylactic doses of digitalis when labor seems imminent, or a few doses of strophanthus at the beginning of labor pains, so as to have the tonus of the heart muscle at its optimum by the time the strain of the second stage is imposed upon it. At periods of acute dilatation, and especially when pulmonary œdema sets in, venesection affords the greatest relief.

If cardiac symptoms disappear the patient may be gradually allowed up and around, but she must be more careful than before, and if signs of a second break in compensation occur, terminating the pregnancy should be seriously considered. This is especially true in cases of mitral stenosis, in which the cardiac accidents of pregnancy are particularly frequent. Women with compensated mitral stenosis may pass through five, six, or seven pregnancies without appearing to be injured by them (Lenhartz), but when cardiac symptoms appear in a case of this disease during the course of a pregnancy it is nearing the danger line, and if these persist in spite of rest and treatment or when compensation is once broken, the danger becomes great.

Fellner's low mortality (21 cases with 1 death) is probably due to the careful practice of Schauta's clinic, which he summarizes in the advice to "terminate pregnancy in cases of mitral stenosis as soon as the slightest signs of broken compensation appear," or in cases in which signs of danger had been present in previous pregnancies.

Case of Mitral Stenosis with Pregnancy and Labor

The danger of disregarding this advice was well illustrated by a patient under the writer's care during the past year. She was a young married woman of twenty-six, and was seen in November, 1907, in the sixth month of pregnancy, complaining of shortness of breath and was quite cyanotic. Her trouble dated from the birth of her first child nine months before, at which time she had evidently received a mild puerperal infection. The veins were rather full, her heart was not enlarged, and at the apex the first sound was snapping and preceded by a slight presystolic rumble. This varied in intensity from time to time. Occasionally a blowing diastolic murmur was heard along the left border of cardiac dulness, but not over the aortic area. The pulse was small and weak, not collapsing, usually regular. There was slight œdema of the shins and ankles. The patient was placed in the hospital, and her condition improved at once, so that within two weeks she was allowed to enter the waiting ward of the obstetrical department. It was then proposed that labor should be induced, but the obstetrical house staff did not regard the case as imperative. She left the hospital contrary to advice and on January 1, 1908, in the seventh month of pregnancy, she was delivered of a healthy premature child. The labor was easy. She insisted upon giving the infant the breast for a couple of weeks, but remained in bed and quiet, suffering

from orthopnœa. This continued in spite of digitalis. Her liver was enlarged, and œdema of the legs gradually set in. She finally returned to the hospital, but never recuperated, and died in June, 1908. The child, which had always been under the care of a district nurse and later in the Thomas Wilson Hospital, also died during the same month.

Termination of Pregnancy.—In an almost exactly similar case Hellendal performed an abortion as soon as the signs of broken compensation were definite, and eight days later resected both tubes to prevent subsequent pregnancy. The patient made a perfect recovery and her life is no longer endangered.

In deciding the question of terminating pregnancy, it must be borne in mind that in from 25 to 40 per cent. of patients with severe heart lesions the pregnancy does not reach term, but premature labor occurs spontaneously owing to partial asphyxia of the fœtus. The placental circulation is slowed, the aeration is poor, and, as Fellner has shown, there is often a large necrotic border about the placenta. This probably results from thrombosis. Moreover, the statistics of the obstetrical clinics, even of Schauta's, are far more favorable than the end results would show. Our own case above mentioned would be classed in such statistics as "improved" at the end of the puerperium and the child as "living"; while, as a matter of fact, both died within six months after the labor. Since most statistics are compiled from the histories of hospitals, where the cases are subsequently lost sight of, it is probable that this represents a very large class of cases. The children are especially delicate, and, even if they survive, succumb more easily to pulmonary and gastro-intestinal infections during the first or second year than do other children.

The inevitably high child mortality and the danger to the mother, especially in mitral stenosis, somewhat lessen the moral responsibility of terminating pregnancy. Moreover, as Weber and Deguy have shown, pregnancy and labor are in themselves causal factors in valvular disease, and especially mitral stenosis, through the occurrence of hemorrhages into the substance of the valves (see Chapter III), or, as in the case of our patient, bring about the recurrence of a slumbering endocarditis, and thus leave the patient worse than before, often with a progressive lesion. When it has been decided to terminate pregnancy, this should be done as soon as possible. The procedure of choice depends upon the severity of the symptoms and the necessity for immediate emptying of the uterus. They have been summed up by Fellner in the following scale: (1) Induction of labor with de Ribes bag or packing the cervix, (2) craniotomy, (3) forceps; (4) version and extraction, (5) Cæsarean section. In general it must be said that the less the operative interference with the physiological course of each stage, the less shock to the patient and the better the end result. On the other hand, each stage of labor is likely to be prolonged in such cases and this must be avoided. When the condition is alarming, the relief should be rapid. Pulmonary œdema is often at once relieved by tapping the fetal membranes, removing the amniotic fluid, and allowing the diaphragm to descend, although the labor then becomes much harder. The patients usually stand the operative interference well. As in other conditions, ether is preferable to chloroform where the heart is diseased.

AORTIC DISEASE IN PREGNANCY.

As aortic disease is comparatively rare in women, it usually receives little mention. It is significant, however, that in Fellner's series there was a very high mortality (60 per cent.) in the fœtus. Newell reports a case in which there was little cardiac discomfort throughout pregnancy, but a hard labor set in. Forceps were used. Collapse and pulmonary œdema ensued, and the mother died four hours after labor. The child died also. This is simply an example of the acute heart failure (probably acute dilatation of the left ventricle with sudden onset of functional mitral insufficiency) so characteristic of aortic insufficiency. Mitral lesions are usually more dangerous than aortic, but they usually give signs of gradual progression. The danger in aortic insufficiency may, as in Newell's case, come on very rapidly and result in the death of the patient.

SUBSEQUENT PRECAUTIONS.

In cases in which dangerous breaks in compensation occur during the course of pregnancy and termination of the latter becomes necessary, as well as in those which reach a natural termination under conditions in which the life of the mother is endangered, measures must be taken to prevent subsequent conception. As Feis points out, the physician's advice to a married woman to absolutely avoid coitus is so rarely followed that for practical purposes it is scarcely worth giving. To rely entirely upon it therefore savors of hypocrisy. Feis believes that in these cases prophylactic measures against conception should be advised. Fellner and Hellendal go one step further. They both advise and practise sterilization of the mother by resection of the tubes, an operation which is not fraught with much danger, and which then relieves her from the sword of Damocles that otherwise hangs over her head.

MATRIMONY AND HEART DISEASE.

The question also arises under what condition may women with heart disease be permitted to marry. As Fellner's statistics show, the danger is not very great. Blacker sums up the facts in the statement that all women with heart lesions will suffer from them sooner or later, and that this period need not be much accelerated by pregnancies. Some writers even go so far as to state that pregnancies do not alter the duration of life at all, but this view is much too optimistic. The best proof, however, that the compensated heart lesion should not be a bar to matrimony is shown by Fellner's statistical proof that six out of every seven heart lesions are not even suspected in the average obstetrical clinic. On the other hand, if compensation is poor, marriage, like any other strain, should of course be forbidden. This again, as Fellner points out, depends as much on sociological as on physical factors, for a woman in poor circumstances may be able to live more quietly and avoid cardiac strain more readily in married life than when supporting herself by her own work. Under such circumstances the patient should be made fully aware of the dangers of conception and coitus. All things being considered, compensated mitral stenosis cannot

be made an exception to these rules, although its presence warrants a certain foreboding in the physician consulted, and should direct his advice toward the side of caution. If compensation has once been broken in a case of mitral stenosis, conception should be forbidden and marriage strongly advised against. The same applies to well-marked chronic myocarditis or nephritic cardiopathy when these can be diagnosed with probability, since they run a more unfavorable course for both mother and fœtus than do the cases of valvular lesions.

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X.

CONGENITAL HEART DISEASE.

DEVELOPMENT OF THE HEART.

In the human heart, as in all other complex anatomical structures, there are many portions whose form and function are obscure and difficult of comprehension when considered only in the light of conditions present in the adult, but which become quite clear when seen in the various stages of their development. A brief consideration of the embryology of the heart will therefore greatly simplify the study of the anatomy. Moreover, it must be borne in mind that occasionally some portion of the adult heart fails to develop beyond the embryonic stage, giving rise to the signs and symptoms of congenital heart disease, and therefore a knowledge of the embryological development is necessary for purposes of practical diagnosis and prognosis as well as for anatomical study.

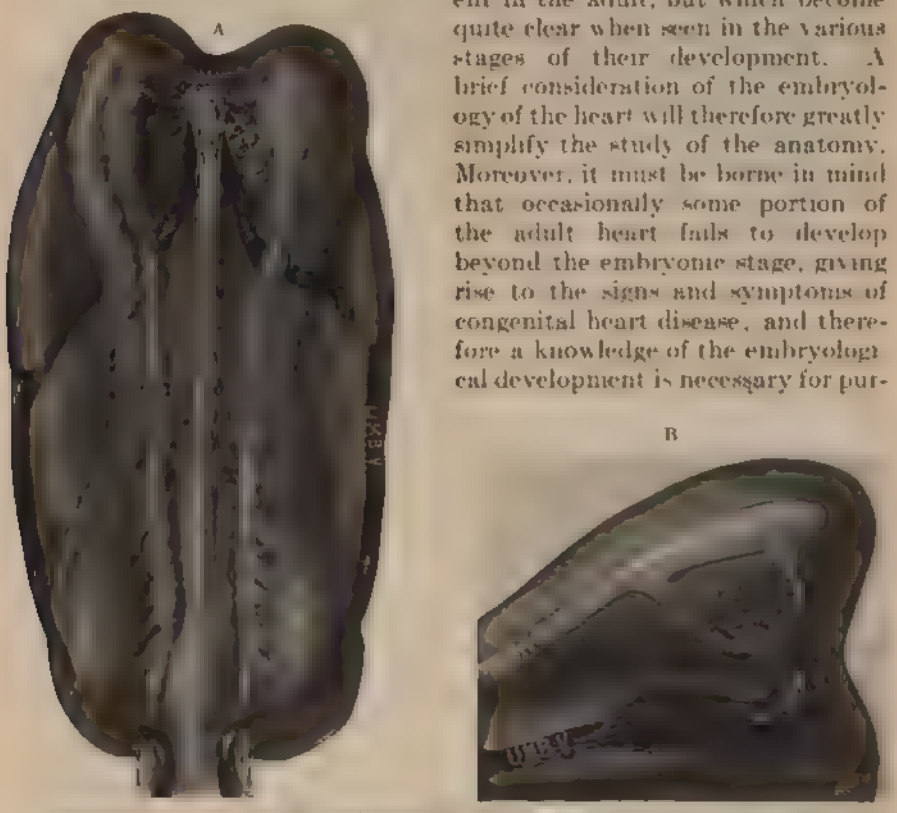

FIG. 236. Very early stage in the development of the human circulatory system. (Drawn from a model of a human embryo 2.5 mm. long, about two weeks after fertilization. From Prof. F. P. Mall's collection. The model was prepared by Mr. W. E. Dandy.) The specimen shows the two aortæ (*Ao*) leading the blood to the placenta (*Pl*), and the two umbilical veins (*UMB*) leading it back to the primitive heart (*H*). The five branchial or aortic arches (*I–V*) which connect the heart with the aortæ are shown also. A, Dorsal aspect. B, Lateral aspect of the head end of the same embryo.

The earliest stage of the circulatory system in the mammalian embryo consists in the formation of a number of small blood-vessels and capillary

plexuses, which arise from the mesoblast over the surface of the yolk-sac. These soon unite to form a definite symmetrical vascular system.

Eternod has carefully described the earliest development of the circulatory system in a human embryo 1.2 mm. long, estimated at about eleven days after fertilization. Another embryo of almost the same age (Fig. 236) has recently been discovered by Dr. Mall and modelled by Mr. W. E. Dandy. In this embryo the venous system is represented almost entirely by the umbilical veins (*Umb V*), which carry the blood that has been aerated in the placenta back to the heart. They follow the border between the embryonic area and the yolk-sac and pass forward to the head end of the embryo. Here (*H*) the two veins unite, and at the union there are given off a second set of vessels consisting of a group of four upon each side, the aortic arches (*A A*), which pass backward more or less parallel to the midline and soon reunite to form a single vessel on each side, the primitive aortæ (*AO*). These two aortæ carry the blood from the embryo proper back on each side of the midline to the numerous ramifications in the placenta, whence, as we have seen, it is returned through the umbilical veins. At this stage the heart is simply a small dilatation of the venous tube, and the blood is propelled by the pulsations of the vessels throughout their entire lengths. There is scarcely a widening of the lumen to mark the site at which the heart will develop,—namely, at the point of union of the two umbilical veins just behind the place where the aortic arches are given off.

Fig. 237.—Human embryo 4 mm. long, about the fourth week after fertilization, showing the further development of the heart and of the branchial or aortic arches (*A A*). Modified from His. The heart has assumed an S shape and is divided into a truncus arteriosus (*TR A*), a single ventricle (*V*), and a single auricle (*AUR*). The inner endothelial cardiac tube, shaded light, is much narrower than the outer muscular tube (*MUS*). On each side the jugular veins (*JUG*) from the head unite with the cardinal veins (*CARD*) from the trunk to form the duct of Cuvier (*D CUV*), which empties into the sinus reuniens (*S R*).

At a little later stage (Fig. 237) we find the heart the form of an S-shaped tube just ventral to the pharynx of the embryo to which it is fixed, and already two dilatations have taken place in the lumen, forming the primitive sacs of the ventricle (*V*) and the auricle (atrium) (*A*). The point of union of the veins (sinus reuniens, *S R*) has been pushed further backward. The umbilical veins have received veins entering from the yolk-sac (vitelline veins) as well as a branch (duct of Cuvier, *D Cuv*) from the body wall on each side. The duct of Cuvier is in turn formed by the union of a branch to the head (jugular vein, *Jug*) and a branch (cardinal vein, *Card*) extending downward along each side of the body wall and giving off branches to the muscle segments. The veins to the intestine arise from the vitelline vein, while the umbilical or omphalomesaraic veins continue as before to carry the blood back to the placenta and yolk-sac.

Anteriorly the arterial portion of the circulatory system may now be observed to be composed of the truncus arteriosus (*Tr A*), a continuation of the ventricle, and four aortic arches each now corresponding to a definite

visceral (or gill) arch of the embryo (*V A*). These branches of the aorta are of great importance, for from them the carotid, axillary, innominate, and pulmonary vessels will develop.

As the embryo grows older (Fig. 240) the heart is still more S-shaped,

Fig. 238.—Heart of an embryo slightly older than that shown in Fig. 237, showing the earliest stages in the formation of two auricular and two ventricular pouches. Drawn from a His model.

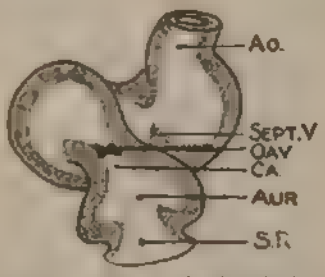

Fig. 239.—A diagram showing the interior of this heart. *AO*, aorta or truncus arteriosus; *SEPT. V*, septum of the ventricles; *OAV*, auriculoventricular orifice; *CA*, canalis auricularis or auriculoventricular channel; *AUR*, auricles; *S.R.*, sinus reuniens or common chamber into which the two venae cavae empty, which corresponds to the sinus venosus of the lower vertebrates.

Fig. 240.—Schema to show the development of the arterial system from out of the primitive aortic arches. A, Schema of the circulatory system at about the same stage as Fig. 237. I, VI, aortic or branchial arches; the fifth branchial arch described by Tandler arises from and anastomoses with the fourth, which is the largest of the branchial arches. *AO AO*, primitive aortae; *PA PA*, rudimentary pulmonary arteries; *V*, primitive common ventricle; *RA LA*, auricular pouches. B, Schema of the adult arterial system derived from the aortic arches. *EXT CAR*, external carotid artery, 3d arch; *INT CAR*, internal carotid artery, connecting bars of first three arches; *COM CAR*, common carotid artery connecting bar between third and fourth arches. A, The aorta is seen to be derived from the fourth branchial arch; the pulmonary arteries arise along the course of the sixth. The ductus arteriosus (Botalli) represents the distal end of the sixth branchial arch. The dotted lines indicate the outline of embryonic arteries which have atrophied.

and at the junction of the two halves of the S a small crescentic infolding of the muscular and endothelial wall has begun to protrude into the cavity of the ventricle (interventricular septum, *Sept. V.*), while the ascending limb of the lower half of the S represents a stenosis in the lumen, the canalis auricularis (*CA.*), whose narrowest part forms a small slit, the ostium auriculoventriculare (atrioventriculare) (*OAV.*).

The development of the interventricular septum continues rapidly (Fig. 241), and also a similar ridge appears running longitudinally along the truncus arteriosus, changing the lumen from circular to U-shaped, each arm of the U being a channel leading to the corresponding half of the ventricle.

The auricular (or atrial) cavity is now also widened into two symmetrical pouches, the right and left auricles, the cavity of which is continuous with the junction of the veins.

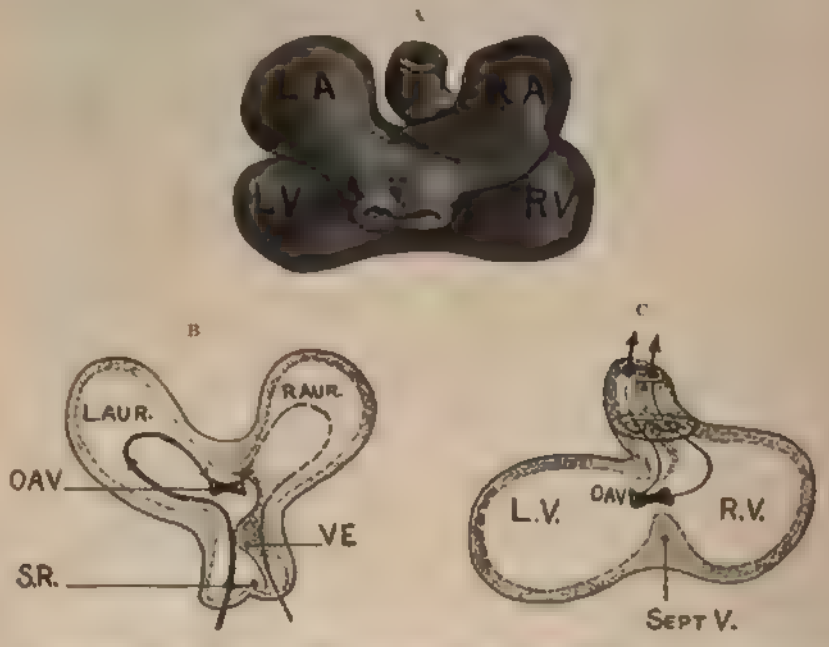

FIG. 241.—A, Heart of slightly older embryo viewed from the dorsal aspect, showing the separation of the aortæ and the pulmonary channels in the truncus arteriosus. Drawn from a His model. B, Diagram of the auricular portion of the same heart. Lettering as in Fig. 239. *VE*, Eustachian valve, separating the sinus from the auricular portion of the heart. The arrows indicate the course of the blood-currents. C, Diagram of the ventricular portion of the same heart, showing the course of the currents through the separate channels of the truncus arteriosus.

The trunks of the veins have already undergone considerable changes, such that the left duct of Cuvier is now atrophied, and most of the blood from the head and upper limb returns to the heart through the right duct of Cuvier, foreshadowing the superior vena cava, while the blood from the placenta returns through the two omphalomesaraic (or omphalomesenteric) veins, which along with anastomoses from the body wall, intestinal tract, and liver will form the inferior vena cava. The junction of the two venæ cavæ forms the sinus reuniens which opens into the auricular cavity. In the wall of the sinus reuniens at this stage there is a longitudinal valve-like fold of endothelium (*VE*, Fig. 241, B), so arranged that blood from the superior vena cava flows over it into the right auricle (atrium), while the blood arriving from the placenta is directed under it into the left auricle.

Very shortly after this stage the most important changes take place in the heart (Fig. 242). The two channels of the truncus arteriosus are now completely separated off from one another, and exist as distinct vessels, the aorta (*Ao*) and the pulmonary artery (*P.A.*), connected with each other at only one point through the ductus arteriosus (*D.A.*). The interventricular septum (septum ventriculorum, *Sept V.*, Fig. 241, C) is now found to be almost completely closed, and the originally single auriculoventricular opening is now divided into two portions (mitral and tricuspid, *Mit.* and *Tric.*) separated by the ingrowth of the septal ridge. In the auricles also great changes have occurred. The greater portion of the sinus reuniens has been drawn into the cavity of the auricle, and exists there as a separate chamber, whose right margin opening into the right auricle is formed by the longitudinal valve (*VE*) (described in connection with the previous stage of development, now known as the valvula venosa dextra, or Eustachian valve). The left wall of the right auricle is formed by the septum auriculorum, which has grown considerably, partly through the gradual ingrowth of the septal ridge and partly by the pushing in of a mass of connective tissue arising from the latter and from the left wall of the sinus reuniens and known as the septum intermedium. The left wall formed in part by the wall of the vein is imperfect, and on the left the cavity extends over to the auricular septum (septum atriorum). This septum has also not completely closed, and the reconstruction (Fig. 243) from a model by Born at this stage shows a double opening between the two auricles. At a later stage (Fig. 245, B) these openings have broken down into one, the foramen ovale, and it is the opinion of Born, in opposition to His, Sr., that the latter structure is of secondary formation and does not arise directly from the primitive interauricular openings, although it performs the same function, — namely, of allowing the blood to pass from the right into the left auricle.

FIG. 242.—Still later stage, showing complete division of the truncus arteriosus into pulmonary artery and aorta. Drawn from a Born model of a rabbit's embryo 10 mm. long. The arrows show the course of the blood-stream. *D.A.*, ductus arteriosus.

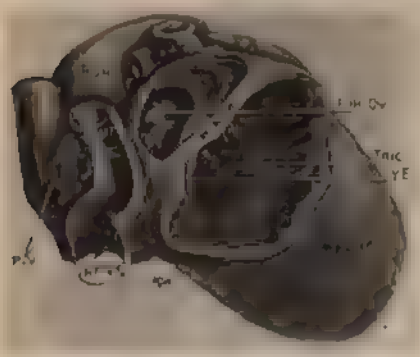

FIG. 243.—Auricular end of the same heart. The blood enters through the superior and inferior vena cava, *SVC* and *IVC*, into the sinus reuniens, *SR*, which is separated from the right auricle, *RAR*, by the longitudinal valve, *VE*, which at this stage forms a large partition between the two cavities. The foramen ovale, *FOR.O.*, connects the sinus reuniens with the right ventricle, *RV*, divided into two parts by a thin lamina formed from the interventricular septum. *TRIT*, tricuspid valve. *PA*, pulmonary vein.

In the later stages a valve-like flap of connective tissue projects over the foramen, allowing the blood to flow only from right to left.

DEVELOPMENT OF THE PERICARDIUM.

The pericardial cavity develops as a part of the original body cavity or cœlom, from which it is separated at a later stage. In the earliest embryos (Fig. 236) the pericardial cavity arises as a small space lined with endothelium, surrounding the blood-vessels on each side of the embryo (Fig. 244, A). These two spaces or cavities unite at the head end of the embryo to form a single pericardial cavity which surrounds the primitive heart. At a slightly later stage the heart and the pericardial

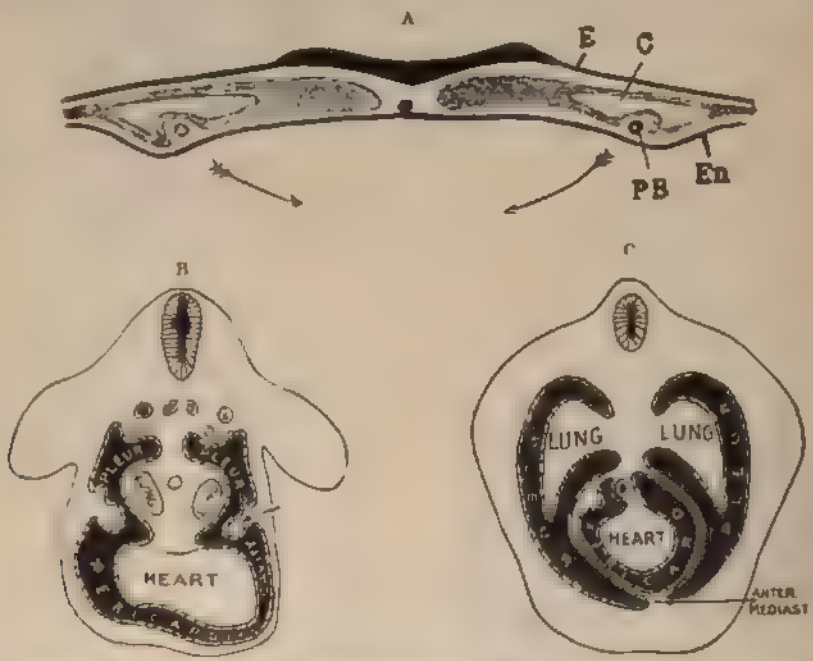

FIG. 244.—Development of the pericardial cavity. A, Earliest stage in the development of the pericardial cavity. (After Robinson.) Embryo corresponding roughly to the stage shown in Fig. 236. *E*, ectoderm; *C*, cœlom; *En*, entoderm; *PB*, primitive blood-vessel. The pericardial cavity is represented by the part of the cœlom present at this level. B, Later stage showing the division of cœlom into pleural and pericardial cavity. Schematic. The arrow points to the channel connecting the two cavities. C, Relations of the pericardium in the adult. (Schematic.) *ANTER. MEDIAST.*, anterior mediastinum.

cavity lie upon the ventral aspect of the pharynx and the pericardial and pleural cavities together form the anterior or cephalic portion of the cœlom.

At a later stage (Fig. 244, B) the heart has grown to fill almost all the ventral portion of the cœlom in its vicinity, and about its contour the connective tissue of the body wall is closing in, as shown by the arrow, beginning to divide the original cœlomic cavity into a pleural and a pericardial portion.

In Fig. 244, C this closure has become complete, and we have represented in rough diagram, the conditions present in the adult chest. The pericardial cavity is completely separated from the pleural cavity, and is lined throughout by a single layer of flat endothelial cells,—the portion growing directly upon the heart called the epicardium, and the portion forming the opposite wall of the pericardial cavity, the pericardium proper.

The pleural cavity has now grown more extensive than before, owing to the growth of the lungs, which have pushed forward along the sides to well in front of the heart and almost to the midline, leaving a narrow pleural cavity between them and the chest wall. Like the pericardial cavity the pleural cavity is lined with endothelium which extends partly over the lung (visceral pleura) and partly along the thoracic wall (parietal pleura). The anterior portion of the visceral pleura passes over the pericardium, from which it is separated only by a very thin mass of connective tissue, occasionally containing fat-cells. The three layers—pleural endothelium, connective tissue, and pericardial endothelium—are so closely fused that together they are generally designated as the pericardium, of which one speaks of the pleural and pericardial surface. The pericardium does not extend quite to the chest wall, while the pleura does so, and ventral to the heart we find a small space filled by connective tissue and known as the anterior mediastinum.

PHYSIOLOGY OF THE FETAL CIRCULATION.

The blood of the fœtus is aerated in the placenta and passes back through the umbilical veins and through the ductus venosus (*D.V.*) to the inferior vena (*I.C.V.*), without passing through the liver. The sinus reuniens has now become part of the main cavity of the auricle, and the inferior vena cava (*I.C.V.*) empties into the latter near the septum ventriculorum. Over its mouth pass the remains of the Eustachian valve (valvula venosa dextra) which directs the blood not into the right auricle but away from it across the right auricle to the limbus fossæ ovalis. According to the views of Galen and Harvey, the blood from the superior vena cava and that from the inferior are mixed in the right auricle before any of the stream passes to the left auricle. Haller and Sabatier, however, believed that no such mixing took place, but that all the blood from the inferior vena cava (aerated blood) passed across to the left auricle, while the blood from the superior vena cava passed down into the right ventricle. Pohlman has recently given an excellent review of the subject. He has investigated it experimentally on the fetal pig's heart by injecting starch granules into the superior vena cava in some living fetal pigs and into the inferior vena cava in others. The hearts were then removed, and the bloods in the two ventricles and auricles were shown to contain the starch granules in equal amounts, confirming the theory of Galen and Harvey. Pohlman introduced capillary glass tubes into the two ventricles and demonstrated that the pressures within them were equal. The blood from the left ventricle passes at first to the innominate and carotid and subclavian arteries, below which the aorta is joined by the ductus arteriosus Botalli. The blood from the

right ventricle passes into the main trunk of the pulmonary artery, from which about one-fifth enters the rami passing to the lungs and about four-fifths passes onward through the ductus arteriosus and enters the descending aorta. As the ductus arteriosus carries a little more blood than the descending aorta, the volume of blood in the aorta is more than doubled and the lumen considerably widened below its entry. The blood below this point goes to the kidneys, the alimentary tract, the bladder, and the lower limbs, and the rest goes on through the umbilical arteries (*Umb.A.*), to be aerated in the placenta and returned as described above.

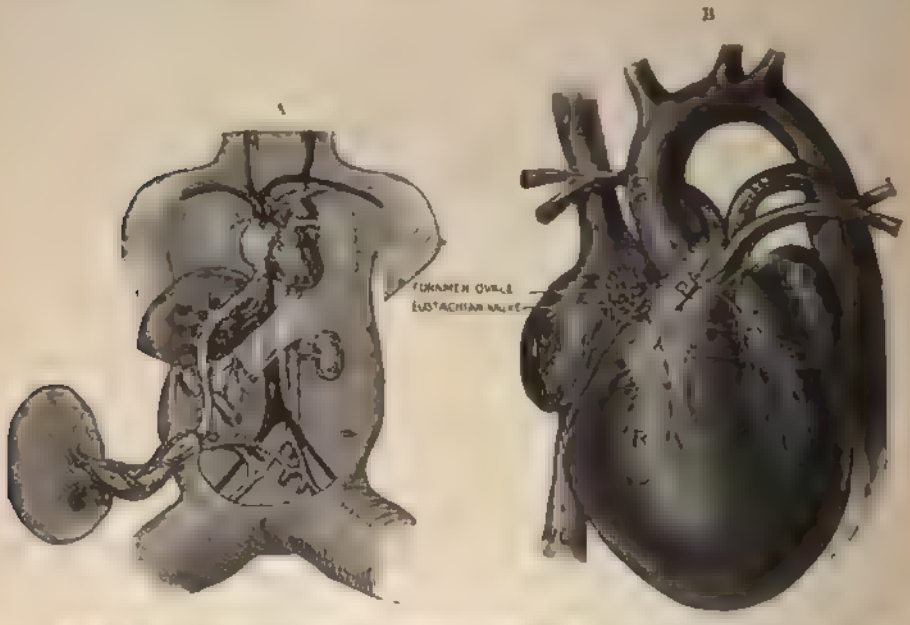

FIG. 245.—A. The circulation in the fetus just before birth. Course of the blood to and from the placenta. Semi-schematic. *Umb.A.* *Umb.V.* umbilical artery and umbilical vein, *Di.* ductus venosus. B. The heart just before birth. The course of the blood stream is indicated by the arrows. *V.S.*, *V.C.I.*, superior and inferior vena cava.

These are the conditions present up to the time of birth. After the first respiration the expansion of the lungs greatly reduces the resistance in the pulmonary circuit, so that it becomes less than that in the aorta, and most of the blood is diverted from the ductus arteriosus into this new channel of low resistance. Hence it persists only a year or so after birth and soon becomes changed into a simple strand of connective tissue.

On the other hand, the pressure in the left auricle becomes greater than that in the right, and the valve of the foramen ovale is therefore kept closed against the septum, and soon becomes organized as a part of the latter.

With the cessation of placental circulation, the ductus venosus loses its physiological importance and soon undergoes atrophy and closure.

CLASSIFICATION OF CONGENITAL HEART LESIONS.

Classifications of congenital heart lesions are difficult, and from a clinical stand-point not always satisfactory. From the anatomical stand-point they may be classified as follows:

I. Malformations about the heart.
   1. Malformations of the chest wall (ectopia cordis).
   2. Malformations of the pericardium.

II. Abnormalities in the position of the heart.
   1. Heart on the right side (dextrocardia or dexiocardia).
   2. Position of all the organs inverted (situs transversus).
   3. Heart situated in the neck (cervical heart).
   4. Heart situated within the peritoneal cavity (abdominal heart).

III. Abnormalities of the valvular orifices.
   1. Pulmonary stenosis or atresia.
   2. Supernumerary or defective cusps of pulmonary valves.
   3. Tricuspid stenosis or insufficiency; malformation of the valve.
   4. Aortic stenosis; atresia of the aorta; malformations of the aortic valve.
   5. Mitral stenosis; malformation of the mitral valve.

IV. Defects in the septa.
   1. Interventricular septum.
      a. In the septum membranaceum.
      b. In the muscular part of septum (below).
   2. Interauricular septum.
      a. Defect or absence of valve of the foramen ovale.
      b. Valve normal but not closed.
      c. Defect between the muscle strands in the lower portion of interauricular septum.

V. Abnormalities in the cavities.
   1. Supernumerary septa
   2. Cor biatriatum triloculare.
   3. Cor biloculare.
   4. Cor biventriculatum triloculare.
   5. Bifid apex.
   6. Double heart.

VI. Deviations of the septum cordis with transposition of vessels.

VII. Persistence of ductus Botalli.

VIII. Abnormalities of the aorta.
   1. Coarctation of the aorta.
      a. Above the ductus arteriosus.
      b. Below the ductus arteriosus.
   2. Hypoplasia of the aorta.
   3. Malformations of the aortic arch.

IX. Abnormalities in the arrangement and formation of the veins.

GENERAL CHARACTERISTICS.

Such a purely anatomical classification, though sufficiently complete, does not furnish a good basis for the study of the cardiac malformations, because it does not take into account the relation of the individual lesions to one another. For, since these lesions are usually produced in groups rather than singly, it is quite as important from a clinical stand-point to recognize these groups and understand their effect upon the circulation as to recognize the individual lesions.

Moreover, as will be seen, the mere clinical manifestations show great similarity in the various lesions, and may be summed up in what may be

termed the "syndrome of congenital heart lesions;" or, in the words of Peacock (1866), "the characteristic symptoms of malformations of the heart—cyanosis (especially from birth), palpitation, dyspnoea, faintings, occasional convulsive attacks and lividity." Moreover, the most common physical sign of many congenital lesions is a loud superficial murmur, most intense in the second and third left interspaces at the sternal margin in both systole and diastole and often heard over the entire precordium and the arteries as well.

ETIOLOGY.

As Lancereaux has well said, "cardiac teratology represents the pathology of intra-uterine life." The chief pathological conditions which affect the development of the fœtus may be classed as

1. Inflammation (fetal endocarditis or myocarditis, the formation of adhesions about the heart or vessels, etc.).
2. Abnormal torsions of the cardiac tube.
3. Underdevelopment of heart or branchial arches.

These processes lead directly to the production of malformations which may be designated as primary congenital lesions, such as stenosis and atresia of the pulmonary artery, transposition of the great vessels, stenosis at the isthmus of the aorta, etc. The presence of these lesions in the fœtus in turn exercises its effect upon the circulation, which alters the course of development and brings about secondary congenital lesions. The developmental mechanics which results in the formation of such groups of lesions is well illustrated in pulmonary stenosis and atresia, the commonest of congenital heart lesions which may be considered as the prototype.

PULMONARY STENOSIS AND ATRESIA.[1]

The commonest of all the primary congenital lesions is pulmonary stenosis, occurring in 254 (68 per cent.) of the 366 cases of congenital heart disease reported by Peacock and by Keith. Two causes have been advanced to explain its occurrence: (1) endocarditis in fetal life; (2) defective development of the pulmonary artery.

1. Bouillaud (1835) ascribed it to endocarditis in fetal life. This theory seems certainly to be applicable to those cases in which the semilunar valves have already formed, but just as in the adult have fused along the lines of closure. This is well shown in Fig. 246 and in a case figured by Peacock. Moreover, a number of cases have been reported in which rheumatism or infectious disease in the pregnant mother has led to the occurrence of endocarditis in the fœtus. On the other hand, it must be borne in mind that in 329 (82.5 per cent.) of 399 cases of pulmonary stenosis collected from the reports of Rauchfuss, Vierordt, and Abbott the interven-

[1] Atresia = (Greek ἀτρέτος (atrêtos = not perforated), from ἀ = not and τρέτος = a boring = absence or extreme constriction of any natural passage or opening of the body.

tricular septum remained incomplete, indicating that the primary lesion had taken place before the time at which the septum had closed (eighth week of embryonic life). As Osler has pointed out, "It is not easy to imagine a fetal endocarditis localized to so small an area as the pulmonary valves must be before the eighth week of fetal life." To this very objec

FIG. 246.—Pulmonary stenosis due to fusion of the cusps. (Drawn from a specimen in the Army Medical Museum, Washington, D. C.) There is also a patent interventricular septum.

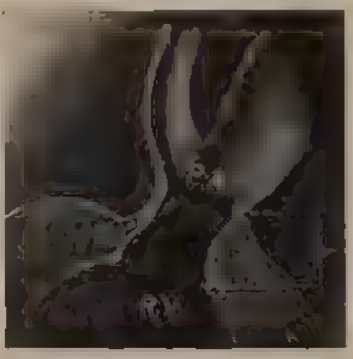

FIG. 247.—Pulmonary stenosis due to a lesion of the infundibulum. (Drawn from a specimen in the Army Medical Museum, Washington, D. C.) *INF.*, infundibular portion of the right ventricle.

tion, however, the advocates of this theory might reply that in very many cases the lesion is by no means confined to the cusps of the valves, but involves the entire infundibulum, over which the endocardium may be thickened and shrivelled (Fig. 247). Nor does it necessarily follow that even though the interventricular septum has once closed it must remain so,

FIG. 248.—Complete pulmonary atresia. (Drawn from a specimen in the Army Medical Museum, Washington, D. C.) The pulmonary artery ends as a blind sac, shown by broken lines, just above the dot which marks the closed pulmonary orifice.

since it may rupture under increased pressure or ulceration may result from the fetal endocarditis. Such phenomena have occasionally been observed (Abbott).

2. The malformation may also arise by "irregular evolution of the branchial arches." Panum has shown that malformations can be produced experimentally in birds by raising the temperature of incubation (fever in the mother), and His believes that at least a consider-

able portion of malformations result from "disturbances of developmental conditions caused by insufficient nourishment, insufficient aeration of the blood, and mechanical causes resulting from malpositions of the uterus, disturbed placental circulation, etc."

It must be recalled that, as shown by Rathke in 1843, the pulmonary artery separates from the rest of the truncus arteriosus about the eighth week of embryonic life, along with the remains of the sixth[1] left branchial arch which forms the ductus arteriosus and the right pulmonary artery which springs directly from this arch (Figs. 240 and 249). The right pulmonary artery, according to Bremer, has sprung in a similar manner from the right fifth branchial arch, but the latter has atrophied and is now represented only by the small segment connecting the right pulmonary artery with the truncus pulmonalis. In the twisting of the cardiac tube and separation of the ventricles the part of the truncus arteriosus corresponding to the pulmonary artery protrudes ventrally while the aortic portion protrudes dorsally. The truncus pulmonalis thus represents the ventral half of the truncus or bulbus arteriosus and springs directly from the infundibulum of the right ventricle (Fig. 249). Stenosis or atresia may therefore take place from arrest of development in three places: 1. In the trunk of the pulmonary artery between the semilunar valve and the point of branching,—*i.e.*, where the pulmonary artery is in close contact with (and perhaps pressed upon by) the main trunk of the aorta. (This condition is represented in a case of Peacock's series.) 2. At the orifice itself (as in Fig. 246), from fusion or stenosis of the valves. 3. Below the valves and within the infundibulum of the right ventricle, as in Fig. 247. In some cases a supernumerary septum may separate the infundibulum from the main cavity of the right ventricle, thus producing the so-called third ventricle. Peacock regarded this structure as representing the condition present in the turtle, but states that "such separation" (into two cavities)

FIG. 249.—Scheme illustrating the genesis of pulmonary stenosis. A, Fusion of the cusps. B, Fetal endocarditis affecting the infundibulum. C, Normal mode of development of the aortic branchial arches. D, Maldevelopment of the sixth branchial arch, leading to pulmonary atresia.

[1] Rathke, His, and the older writers speak of the last branchial arch from which the pulmonary artery arises as the fifth branchial arch, but Tandler (Zur Entwicklungsgeschichte der Kopfarterien bei den Mammalia. Morphol. Jahrb. Leipz. 1902, xxx, 275) has recently shown that a small rudimentary arch is present upon the same stem with the fourth. He terms this small arch (which plays no important rôle in development) the fifth, and the pulmonary arch accordingly becomes the sixth.

"may be produced in different ways. It may depend simply on undue development of the ordinary muscular bands, or on this in conjunction with thickening of the endocardium or subjacent fibrous tissue." Recently Arthur Keith, of London, has revived Peacock's idea that this is the portion of the heart which is homologous with the bulbus cordis of the lower animals, and which, as Greil has shown, becomes incorporated into the substance of the ventricle (infundibulum) just as the sinus is swallowed up by the auricle. Keith believes that the period during which this is taking place represents the crucial epoch in the production of malformations.[1]

Certain it is that most fetal lesions arise about the time when the pulmonary artery and the aorta and the remnants of the branchial arches are taking their final form, the interventricular septum is becoming complete, and the original portions of the branchial arches are disappearing, — i.e., between the fourth and the eighth week of fetal life.

SECONDARY MALFORMATIONS.

As has been stated above, stenosis of the pulmonary orifice results in stasis within the right ventricle, and the blood is forced to take a new channel.

Patent Interventricular Septum with Pulmonary Stenosis.—In 80 per cent. of the cases of pulmonary stenosis the interventricular septum is still open, and the blood is forced through the open septum and passes up through the aorta. As the condition is a permanent one, the current through the septum continues and its closure is prevented (stasis theory of William Hunter and Kussmaul). In rare cases, and especially those in which the stasis appears at a very early stage, the blood current eddies through and keeps open a passage between the muscle strands at the base, in contrast to the usual defect at the septum membranaceum. This opening at the base of the septum is often accompanied by defects in the mitral or tricuspid valves.

Dextroversion (Rechtslage) of the Aorta. Moreover, the pressure upon the septum tends to deflect it toward the left and still further enlarge the septal opening (Figs. 248 and 250).

In most cases the deflection of the septum to the left is so great that the aorta comes to lie in the axis of the right ventricle. The cavities thus come to form an inverted Y whose arms are formed by the ventricles and whose shaft is the aorta. Since the shaft is inclined to the right, this gives the appearance as though the aorta arose directly from the right ventricle (Rechtslage = dextroversion of the aorta). This condition is present in the majority of the cases reported by Abbott especially in those in which there is complete atresia of the pulmonary artery (Fig. 248).

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[1] It is possible that in some cases like those figured by Keith, the rudimentary septa represent endocardial pockets upon the wall of the ventricle. Schmincke, Endokardiale Taschenbildung bei Aortenmsuffizienz, Arch. f. path. Anat. etc. Berl., 1908, exev. 50, has shown that similar pockets may be formed in the left ventricle by the impact of a regurgitant blood stream.

Open Ductus Botalli.—When the stenosis reaches a considerable grade, much of the blood that reaches the lungs must pass to them from the aorta back through the ductus arteriosus (Botalli) (Fig. 256), which is therefore forced to remain open after birth.

FIG. 250.—Currents and lines of force in the embryonic heart which result from pulmonary stenosis and tend to produce patency of the septa and of the ductus arteriosus. *A*, Ventricular end of the fetal heart before the eighth week of embryonic life. *B*, Auricular end of the fetal heart at the same stage. *P*, pulmonary artery, *D*, ductus arteriosus Botalli, *R A*, right auricle, *R V*, right ventricle, *L A*, left auricle, *L V*, left ventricle, *F O*, foramen ovale. The large arrows indicate blood currents—the black within the ventricles, the white those within the auricles. The small arrows indicate the forces tending to dilate the heart and to deflect the septa. In cases of extreme grades of pulmonary stenosis the current in the ductus arteriosus flows from aorta to pulmonary artery, instead of in the reverse direction.

Open Foramen Ovale.—If the intraventricular septum has closed before the pulmonary stenosis has occurred, the resulting stasis causes a rise of pressure in the right auricle, and the path of least resistance to blood flow is through the foramen ovale to the left auricle. The stream in this direction is therefore larger than usual and prevents the initial sclerosis about the foramen, or even preserves a channel in the lower part of the septum, so that sometimes (as in Fig. 250) the valve of the foramen may close and a breach through this portion of the septum still remain patent.

Three-chambered Heart (Cor biatriatum triloculare).—When the atresia is complete and the intraventricular septum is closed, the right ventricle becomes converted into a blind sac into which no more blood can enter. The tricuspid orifice thus falls into disuse, and the valve undergoes stenosis and atresia until it is completely closed. The cavity of the right ventricle remains only as a small blind sac in the wall of the left, from which the aorta arises, cor biatriatum triloculare (Fig. 251).

Lesions of the Peripheral Vessels.—The abnormalities in structure secondary to congenital heart diseases are by no means confined to the heart, but especially involve the finer ramifications of the vessels. Recent experimental investigations throw much light upon the distribution and formation of these abnormalities in a manner which is of great practical importance.

J. Loeb in 1893 was the first to demonstrate experimentally the effect of injury to the heart upon development. He poisoned the hearts of fish (Fundulus embryos) by temporary immersion in 1.5 per cent. KCl solution and found that, though the hearts of such embryos did not beat at all, nevertheless these embryos reached adult stage and differed from normal fish chiefly in the irregular structure of their blood-vessels. Knower, working with frog tadpoles, has recently confirmed Loeb's observations, but studied the changes

in more detail, and has found that after mechanical or chemical (acetone-chloroform) injury to the heart the embryos usually become very œdematous and are less advanced than the controls. These embryos, according to Mall, are very similar to the œdematous moles frequently met with in gynæcological practice. Knower also found that the development of the brain, intestines, liver, and pancreas is retarded, "both arteries and veins are very much distended, and follow very irregular courses. In most cases the first precapillary loops are represented by large sinuses, . . . but there is a notable absence of capillaries in the fin. The smaller vessels do not push out nor form characteristic plexuses. Their development is inhibited. The weaker the heart-beat in fact the less does the blood flow outward from the larger vessels and precapillary loops." Similar changes had already been described by Panum and Dareste in chick embryos, by Stockard upon fish embryos poisoned with lithium, and by Bardeen upon toads which had been fertilized with sperm previously exposed to the action of X-rays. Knower also notes that similar malformations are common in frogs at the end of the breeding season (when the sperm may well be weakened).

The secondary changes in man, outside of the heart, are quite homologous with those in animals. These are especially underdevelopment in stature and in intelligence and the occurrence of malformations of the arterioles and venules. Just as in Knower's frogs, there is a dilatation and irregularity of venules often in the skin, viscera, and retina (Fig. 253), from which hemorrhages frequently take place. Thickening and clubbing of the ends of the fingers (clubbed fingers, Fig. 254) also take place, from proliferation of the connective tissue as a result of the venous stasis.

FIG. 251.—Three-chambered heart of Ustitalanus trilocularis produced by complete removal of the pulmonary and tricuspid valves. Preparation in museum of the Army Medical Museum, Washington, D. C. *RVD* rudimentary cavity corresponding to the right ventricle. *LV* (transparent) The arrows indicate the course of the blood stream.

PATHOLOGICAL PHYSIOLOGY.

The effect of pulmonary stenosis upon the mechanics of the circulation in the adult is very marked. In the first place it brings about a fall in blood-pressure (both arterial and venous) in the pulmonary artery and in the lungs (Fig. 252), and consequently a corresponding secondary lowering of pressure in the aorta.

The extent to which other areas of the circulatory system are affected depends as much upon the correlated defects as upon the stenosis itself. If the stenosis is the only lesion, it produces a fall of pressure in the pulmonary artery, a rise of pressure (from stasis) in the pulmonary veins, and a marked increase in pressure within the right ventricle, like that which Lüderitz found in the left in aortic stenosis (Fig. 252, light broken line). This always leads to hypertrophy of the right ventricle and right auricle, and usually to the signs of congenital venous congestion to be described later.

Between these two grades of severity there exist all stages of cardiac insufficiency, the most important being the overloading or weakening of the right ventricle, which leads to transitory venous stasis, tricuspid insufficiency, and cyanosis. The pressure in the pulmonary vein and left auricle

is by virtue of the pulmonary stenosis lower than usual, while that in the right auricle is for the same reason higher. Accordingly the tendency is for venous blood to pass into the left auricle and ventricle in diastole to a much greater degree than when the pulmonary orifice is normal, and hence to cause a greater tendency to cyanosis and dyspnœa than in the uncomplicated patent foramen ovale.

When the foramen ovale is patent but the septum ventriculorum closed (12 per cent. of Abbott's cases), the effect upon the circulation varies. Owing to the pulmonary stenosis, the path of least resistance is through the open foramen ovale into the left auricle without passing through the lungs, and much blood may circulate in this way. Whether or not this gives rise to cyanosis depends upon the actual amount entering the lungs through the pulmonary artery. Under ordinary circumstances this may

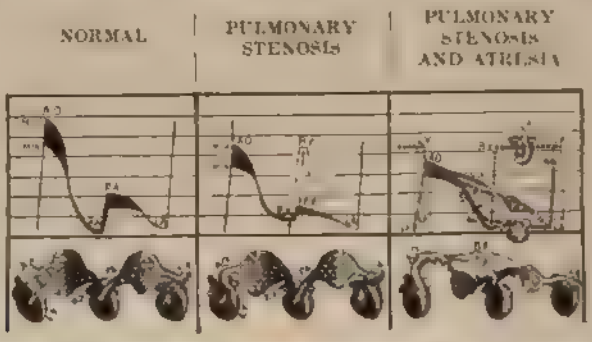
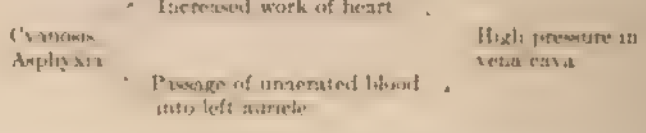


FIG. 252.—Diagram of the circulation in pulmonary stenosis and atresia. Simple pulmonary stenosis. The arrows show the force of pressure in the aorta and pulmonary artery and the rise of pressure in the vena cava and right auricle. The broken line indicates the high intraventricular pressure in the right ventricle. Pulmonary atresia with patent interventricular septum. *ST* patent ductus arteriosus. *DA* and patent foramen ovale. *FO*. The blood current passing through the patent interventricular septum, indicated by the heavy broken line, the light broken lines indicate intraventricular pressure. The shaded curves indicate mixed blood. *BB* bronchial arteries. The relation of the new channels to the other arteries is shown in the diagram below.

keep enough blood aerated to avoid cyanosis, but in exercise or exertion when more CO_2 is produced, this excess may show in the patient's color. Moreover, the venous pressure may rise until the pressure in the right auricle still further exceeds that in the left, and thus a larger proportion of this non-aerated blood enters the left side of the heart, giving rise to the vicious circle of the open foramen ovale (Fig. 250, B).



SYMPTOMS

The classical picture of pulmonary stenosis and especially of pulmonary atresia is the "morbus cœruleus" or "blue sickness," as which it has been known since the time of Senac (1749). The patient is usually a small child

or youth below the normal size and intelligence. He is said to have been blue at birth (as in 74 of Peacock's 101 cases), or to have become so during the first year or two of life (as in almost all of Peacock's other cases). He has suffered from cough most of his life, as well as shortness of breath. The latter becomes extreme or may come on in severe paroxysms after exertion. During these attacks of dyspnœa, the patient may become extremely blue or even black in the face, and they may end in a fainting spell or an epileptiform convulsion (due to venous stasis and cerebral ischæmia). He may also have frequent headaches. His hands and feet are usually cold (venous stasis). He is subject to frequent bleedings from the nose, mouth, intestines, or other mucous membranes (due to congestion in dilated venules), which may even suggest the diagnosis of hæmophilia.

On the other hand, persons with a considerable grade of congenital pulmonary stenosis may remain free from symptoms and even perform heavy work, as in the case quoted by Peacock of a man of forty-four who worked as a navigator until six weeks before his death. Such cases, however, constitute only a small percentage of every series.

Pulmonary Stenosis with Patent Interventricular Septum.—However, when the interventricular septum is defective the condition is entirely different. The lowered pressure in the pulmonary artery usually continues unless fully compensated by the anastomotic circulation. The right ventricle hypertrophies until it equals or even exceeds the left in thickness, and, owing to the dextroversion of the aorta, sends its large quota of blood into the aorta. The pressure in the systemic veins, therefore, depends not upon the pressure within the right ventricle but upon the ability of the right ventricle to force the blood onward and prevent it from accumulating in the veins. The presence, extent, or absence of symptoms, on the other hand, depends upon the aeration of blood in the lungs. Peacock well says that "in cases of this description the open state of the foramen ovale and the imperfection in the ventricular septum, so far from adding to the danger, really afford the means of relief to the overcharged right auricle and ventricle without which life could not be prolonged for any considerable period." That the outlook in cases where defective septa accompany the pulmonary stenosis is graver than in cases where the stenosis occurs alone is due merely to the fact that in the latter case the lesion is usually formed late in fetal life, and hence is comparatively mild, or, if formed early, it is too slight to give rise to the stasis which keeps the septa open.

PHYSICAL SIGNS.

The patients, usually children, are of stunted growth, with eyes watery, veins of forehead, face, and arms large, very numerous, and anastomosing frequently.

Cyanosis.—So striking and so frequent is the occurrence of intense cyanosis in congenital heart disease that this term has become almost synonymous with the "morbus cæruleus" (blue sickness) described by Senac.

The patients may be persistently livid or the cyanosis may be present only at times of exertion or ill health. It may then come in attacks asso-

ciated with dyspnœa and sometimes convulsions. The patients may become quite black in the face and may remain so for some time.

The mode of origin of the cyanosis in congenital heart disease is a matter not only of scientific interest but of the greatest practical importance in diagnosis and prognosis. Theories. 1. Mixture of venous with arterial blood. Senac (1749), William Hunter, Forget, Meckel, Corvisart, Gintrac, Favre, Paget, and others supposed that the cyanosis was due to the passage of venous blood directly into the left auricle or ventricle through the open foramen ovale or interventricular septum. While this may play a rôle in some or indeed in most cases, Moreton Stillé (1844) has shown "that complete admixture of the blood may take place without cyanosis." This is conclusively demonstrated by the two following cases:

"First. Foramen ovale open, pulmonary artery arose from both ventricles, gave off pulmonary branches, and formed the aorta descendens. The aorta gave off the branches to the head and upper extremities and joined the pulmonary artery by the ductus arteriosus. No cyanosis. Age eight months.

"Second.—Heart with two cavities, aorta and pulmonary arising from the ventricle. No cyanosis. Age eleven days."

Numerous other observations in the literature have confirmed Stillé's contention. On the other hand, when there is mixing of venous and arterial blood, a relatively slight stasis or cardiac weakness from overstrain or disease may bring about intense cyanosis, which would not occur in persons with healthy hearts.

2. The second theory, proposed by Morgagni (1761) and subsequently advocated by Louis, Bouillaud, Valleix, Hasse, Stillé and Rokitansky, was that owing to the pulmonary stenosis there was stasis in the systemic veins, and that the cyanosis resulted from that factor only. To this theory Grancher adds the fact that the capillaries and precapillaries are already markedly dilated (for reasons given above on page 434) and that in these dilated capillaries slight stasis brings about marked cyanosis.

Another factor, to which attention has been called by Vaquez, Osler and others, is that in such cases cyanosis is usually accompanied by intense polycythæmia, and the increase in the amount of the CO_2 hæmoglobin intensifies the cyanosis which might otherwise be present in moderate degree.

None of these theories, however, explains the absence of cyanosis in cases where all the venous blood passes into the aorta. In these cases the ventricles are strongly exerting both their suction-pump and force-pump action, so that blood does not accumulate in the veins. On the other hand, the pressure in the aorta (ranging from 90 to 120 mm. Hg) is about three times as high as the pressure normally present in the pulmonary artery, and hence is capable of forcing a very large amount of blood through the wide bronchial arteries or open ductus arteriosus (Botalli) to the lungs.

Whether admixture of venous blood will or will not produce cyanosis depends largely upon the amount of CO_2 which the abnormal pulmonary circulation can take care of, and which in most cases is more limited than in the normal individual. When excessive exercise, strain, or cardiac weakening causes an abnormal increase of CO_2, cyanosis makes its appearance, and owing to the congenital dilatation of the capillaries the cyanotic effect is magnified.

The cases in which cyanosis occurs in spells are probably examples of transitory venous stasis (in congenitally enlarged capillaries and capillary plexus). In cases with open foramen ovale stasis in the systemic veins and right heart will divert an abnormally large amount of venous blood through the foramen ovale (Fig. 230).

The head occasionally shows signs of other abnormalities in form. There may be deficient formation of bones of skull, abnormalities in the form of the ears, hare-lip, cleft palate, etc.

Vascular Changes in the Retina.—Marked changes in the vessels of the retina, seen upon examination with the ophthalmoscope, were first reported by Knapp in 1861. In cases with marked cyanosis elsewhere there are often irregularities in the lumina of arteries and veins, which are tortuous ("resembling large angle-worms," Posey) and in some places very wide (twice as wide as normal), in others very narrow. "Both veins

and arteries become much darker than normal, the former assuming a deep violet color, while the arteries resemble normal veins. The peripheral twigs of the retinal vessels are distended, and vessels which are usually invisible may be seen over the entire fundus. . . . . Small hemorrhages are of frequent occurrence" (Posey) (Fig. 253). In doubtful cases this feature may be of great diagnostic value, but it can be expected to occur only in those cases in which there is a considerable degree of cyanosis and in which the prenatal slowing of circulation has probably been marked.

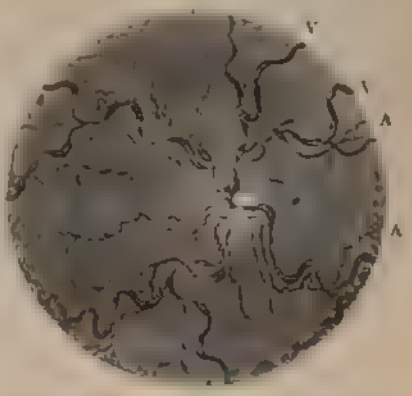


FIG. 253.—Dilatation and irregularity of the retinal vessels. *V*, vein; *A*, artery. (After Posey.)

Clubbed Fingers (Hippocratic Fingers). The ends of the fingers undergo peculiar changes (clubbed fingers, Hippocratic fingers) (Fig. 254), which also occur in chronic pulmonary diseases, tuberculosis, chronic cardiac disease, especially in children and in conditions producing long-continued local or general venous stasis (Ebstein). The change is confined to the pulp of the finger, which is thicker than normal and broadest near the tip, and tapers in a proximal instead of a distal direction. The nails are very convex in both longitudinal and transverse diameters. They are usually cyanotic. The form of the bones is practically unchanged. E. Ebstein has collected a large number of observations which prove that clubbing of the fingers and toes results from chronic passive congestion either general or local (from pressure on veins). The earlier in intra-uterine or in extra-uterine life that the congestion occurs and the longer is its duration the more marked is the clubbing.

FIG. 254.—Clubbed fingers.

The occurrence of clubbed fingers is dependent on almost the same factors as cyanosis, and the two features usually occur together or are both absent.

Cardiac Signs.—The precordium almost always bulges, and the wavy systolic impulse over the precordium due to systole of the right ventricle is usually seen. A systolic impulse at the apex may or may not be present. The area of cardiac dulness is usually enlarged to both right and left. On palpation a rough systolic thrill is felt over the pulmonary area, from which it is transmitted diagonally upward toward the left clavicle and downward over the precordium (Fig. 255). In cases in which there is a defect in the septum ventriculorum, this thrill is also intensified over the third and fourth left interspaces near the sternal margin.

The characteristic sign of pulmonary stenosis on auscultation is a systolic murmur accompanying the above-mentioned thrill and following

the first sound, loudest over the pulmonary area or just beyond it in the second left interspace. In sharp contrast to the murmur of aortic stenosis, it is transmitted upward and to the left. It is also heard over the precordium, but, unless the interventricular septum or ductus arteriosus (Botalli) is open, it is not usually transmitted to the systemic arteries. The second pulmonic sound is either absent or suppressed in spite of the respiratory distress. However, it is noteworthy that in those cases in which there is uniform pulmonary atresia extending over one or two cm. and where the lesion is actually most severe, the murmur may be entirely lacking. The same applies, of course, to complete obliteration of the pulmonary artery. Needless to say, the imperfectly formed valves may be insufficient and a diastolic murmur due to regurgitation may also be present. However, a murmur of this type is most frequently due to the defect in the interventricular septum (see page 444).

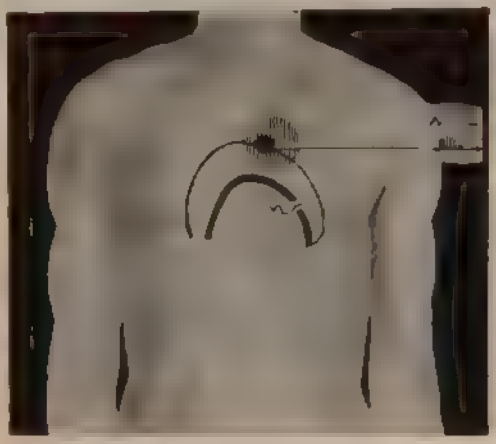


FIG. 255.—Distribution of the pulmonary systolic murmur of pulmonary stenosis.

Open foramen ovale, when present, rarely gives characteristic signs, but occasionally may be diagnosed from a presystolic murmur heard at the base. The signs due to an open ductus arteriosus Botalli are so similar to those of pulmonary stenosis that even in typical cases it is almost impossible to diagnose in the presence of the latter. The murmur from the former is more frequently heard at the back to the left of the third and fourth dorsal vertebræ with every marked inspiratory accentuation and expiratory diminution (François-Franck).

The chest is usually poorly expanded, often pigeon-breasted.[1] Harrison's grooves are often prominent. Signs of phthisis (areas of dulness, tubular breathing, increased vocal fremitus and râles) are very common, especially at the apices (80 per cent. of Abbott's cases), in cases which have passed the age of infancy, and tubercle bacilli are frequently found in the sputum.

FIG. 256.—Direction of blood-streams and propagation of murmurs accompanying defect in the interventricular septum, pulmonary stenosis and open ductus arteriosus Botalli. *DEF SEPT* defect in the interventricular septum. *PULM* pulmonary stenosis. *PAT DUCT* patent ductus arteriosus Botalli. 1, 2, 3, 4, 5 represent the corresponding ribs.

[1] This flatness of the chest may perhaps be of reflex origin, since F. Kaudern (Ueber einige Experimente zur Lehre von der cardialen Dyspnoe, Wien. klin. Wchnschr., 1901), under v. Basch's direction, has shown that the diaphragm rises when the blood flow through the lungs is diminished.

Condition of Other Organs.—The abdomen is often very full, the liver and spleen enlarged, especially in cases with cyanosis and venous stasis. The genitalia are usually underdeveloped.

The blood count usually ranges between 6,000,000 and 9,000,000, the hæmoglobin between 110 and 130 per cent.

There are often albumin and casts in the urine, which is frequently scanty. Occasionally there is blood from the dilated capillaries.

DIAGNOSIS.

As stated by Rauchfuss in 1878, the diagnosis of congenital pulmonary stenosis can usually be made from the following symptom complex: "Cyanosis, from birth or following signs of cardiac affection which were then present; signs of dilatation and hypertrophy of the right auricle and ventricle; systolic murmur and thrill over the conus arteriosus and pulmonary artery, not transmitted to the carotid arteries." He admits, however, as do all subsequent authors, that the exact diagnosis of the secondary lesions *intra vitam* is almost impossible, owing to the multiplicity of the lesions which may occur and the fact that so many of the signs overlap one another.

TREATMENT AND PROGNOSIS.

As regards prognosis statistics vary considerably. Of Stoelker's 53 cases 32 died at birth, 12 during the first year, and 11 during the first decade. Only 4 reached the fourth decade.

The age of death in Abbott's series was as follows:

Age at death.	Pulmonary Stenosis.			Pulmonary Atresia		
	V. S. closed.	F. O. closed, defect V. S.	F. O. patent, defect V. S.	V. S. closed.	F. O. closed, defect V. S.	F. O. patent, defect V. S.
Before 1 year	0	4	3*	6	2	10†
1–7	2	16	3	0	3	0
7–14	4	5	4	0	0	0
14–20	3	8	5	0	0	0
20–28	6	3	0	0	0	0
28–45	1	0	0	0	0	0
	61	36	20	6	5	10

* 9.7 per cent. † 78 per cent.

One can hardly fail to be struck by the contrast between the cases of pulmonary atresia and pulmonary stenosis, since 78 per cent. of the former die in the first year, while this is the case in only 9.7 per cent. of the latter. Even of these only 36 per cent. survived the age of puberty and only one reached middle age.

In the individual case the physician may be guided by the intensity of the symptoms even more than by the physical signs, severe symptoms, as a rule, portending an early death. When the symptoms in early youth

are comparatively mild, the prognosis is a little better, but an early death from phthisis or acute endocarditis is always to be feared, even when the heart failure is less intense. It is, therefore, most important, as Peacock suggested: (1) to keep the patient warm by both warm clothing and sojourn in a balmy climate; (2) to keep him leading a quiet life on a diet of nourishing but easily digested food. For paroxysms of dyspnœa and distress free purgation should be resorted to. Venesection, which was recommended by Peacock, though indicated by both the venous stasis and the high viscosity of the blood, is a dangerous procedure and should be used only as a last resort, for the coagulation of the blood in these cases is often retarded. Before performing it the coagulability should always be determined.

The general cardiac stimulants, such as digitalis and strychnine, are rarely of much value, since in most cases the heart has already reached the maximum of its power and cannot be stimulated much further. Vasodilation from amyl nitrite and nitroglycerin may sometimes help, and Peacock recommends the use of warm baths or mustard baths, especially for the convulsions of children.

DEFECTS IN THE INTERVENTRICULAR SEPTUM

OCCURRENCE AND PATHOGENESIS.

As has been stated above, defects in the interventricular septum are usually with and secondary to other malformations, this being the case in 117 (78 per cent.) of 149 cases studied by Abbott. Pulmonary stenosis or atresia was present in 75 cases (58 per cent.). In only 24 cases (16.1 per cent.) were there no other abnormalities.

The circulatory mechanism which keeps the septum from closing in the presence of pulmonary stenosis has been discussed above under the latter condition. In the other cases, in which Abbott classes it as a "secondary lesion," the mechanism is similar.

In the uncomplicated cases, however, the causation is more obscure. In a few cases it is accounted for by fetal endocarditis affecting the septum interpositum before the septum membranaceum has formed. In other cases the septum membranaceum does not form completely. After birth, when the pressure in the left ventricle rises high above that in the right, the rush of blood from the left ventricle into the right may push the septum along with it and may cause it to protrude as a funnel into the right ventricle (Tate, Hebb). In still other cases the septum forms and protrudes as an aneurism of the septum. This aneurism may rupture later and give rise to the defect.

Some cases of apparent defect in the septum are due to ulcerative septal endocarditis, but these are probably few. Trauma may produce a similar effect in adult life. McOscar and Voelcker report the case of a man who was run over by a wagon. Rupture of the interventricular septum resulted and the patient died eight days afterwards. Reiss states that pulmonary tuberculosis has been found in every adult in his series, but this is by no means always the case.

PATHOLOGICAL PHYSIOLOGY.

When the defect in the septum is secondary to a severe pulmonary stenosis or atresia, as has been seen, its effect is to allow blood to pass from the right ventricle into the left, and under any circumstances this is the case during fetal life.

When there is no such stenosis, however, and the strength of the left ventricle increases after birth, the current passes in the reverse direction and aerated blood passes from the left ventricle into the right. The effect upon the work of the former is consequently about the same as that of a leak at the mitral valve; intraventricular pressure is lowered, and the systolic output must be increased in order to maintain the circulation. The left ventricle consequently hypertrophies as a result of the strain; the right ventricle hypertrophies also as a result of the increase in the blood forced into it. The extent of hypertrophy of the latter chamber depends largely upon the size of the opening. As the right ventricle hypertrophies and pressure in the right ventricle increases, the leakage diminishes, so that the effect of the lesion tends to correct itself, on the other hand, the pressure in the pulmonary artery increases. But since the ordinary resistance in the pulmonary circulation is much less than that in the systemic, when the forces of both ventricles approximate one another, the effect on the pulmonary circulation is the same as though the left ventricle became weaker and the right remained unchanged. Pulmonary engorgements may, therefore, result, with consequent dyspnœa. In most cases, however, the hypertrophy does not reach this point, and it is only when the heart is stimulated by effort or exercise that pulmonary engorgement sets in.

SYMPTOMS.

In considering the symptoms and signs of defects of the interventricular septum, one must differentiate sharply between those cases in which the condition exists alone and those in which it is secondary to other lesions. In the latter case the manifestations of the primary condition may predominate; and these are discussed in the corresponding sections.

The symptoms from simple defect in the interventricular septum are few, and, as a rule, are confined to more or less weakness, dyspnœa, and palpitation, rather than the extensive symptom complex met with in pulmonary stenosis.

PHYSICAL SIGNS.

In marked contrast to pulmonary stenosis, marked cyanosis is not one of the signs of uncomplicated defect in the interventricular septum, since there is, as a rule, no stasis in the veins and the abnormal blood stream flows from left ventricle into the right. Cyanosis may occur, however, as the result of a cardiac overstrain, just as in any other condition of cardiac weakness, but is not abnormally intense. The fingers are, as a rule, not clubbed. Over the precordium and epigastrium there is usually violent systolic retraction, produced by the hypertrophied right ventricle. There may be violent systolic pulsation of the conus arteriosus in the second left interspace. The area of dulness may be enlarged to both

right and left, or there may be no change from the normal. There is almost always a well-marked systolic thrill over the third left interspace near the sternal margin.

Auscultation reveals the presence of a murmur which was first described by Roger in 1879 in the following words:

"It is in general remarkably intense; its maximum is not at the apex (as in alterations of the auriculoventricular orifices), nor at the right base (as in aortic stenosis), nor at the left base (as in pulmonary stenosis). This maximum is at the upper third of the precordial region and is median like the ventricular septum itself. It is single and very prolonged, commencing with systole and replacing the two normal sounds. It is fixed without propagation in the large vessels as is the case with aortic or pulmonary stenosis, and decreases in intensity equally in all directions as one passes away from this central point. The murmur corresponds with a very extensive thrill which exactly coincides with it. The murmur does not change in the course of years."

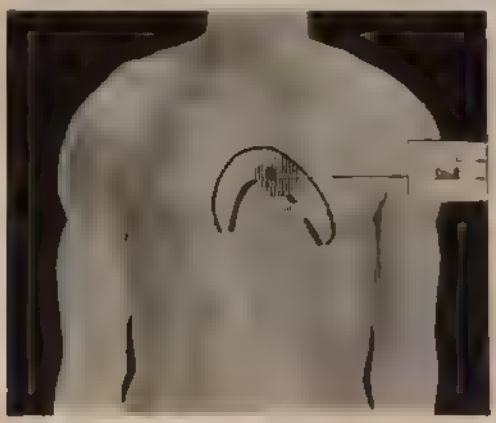


Fig. 257.—Distribution and character of the murmur due to a patent interventricular septum (Roger's murmur).

However, all writers do not agree with Roger. Cadet de Gassicourt, Potain, and Reiss claim that it occurs during systole only; while in some cases, especially where the septal defect is a large one, it is totally absent (Bennetz). The murmur is sometimes transmitted to the carotid arteries, though it is always loudest over the precordium. The second pulmonary sound is accentuated.

The pulse may be small and weak, or, as in the case of McOscar and Voelcker, collapsing. The blood-pressure is usually low.

Case of Patent Septum Ventriculorum

The following notes were obtained from a case admitted to Prof. Barker's service in the private wards of the Johns Hopkins Hospital.

The patient was a married man, a scientist of some note, aged 59. As a child he had been subject to bad dreams and disturbed sleep and became short of breath on slight exertion. This shortness of breath on exertion followed through life, but in spite of the ordinary diseases of childhood, three mild attacks of typhoid fever, and continued use of tobacco, alcohol, and strong coffee, he was able to lead an active life until past middle age.

For six weeks before admission to the hospital he has been very weak and has been troubled with nocturnal dyspnœa, though these symptoms are probably referable to his renal rather than to his cardiac changes. Swelling of the feet set in a few days before admission.

On Feb. 15, 1908, Dr. Barker made the following note on his cardiac condition: The radials are thickened, the blood-pressure is high, there is a blowing systolic murmur at the apex, the aortic second sound is fairly loud, the pulmonic second very loud. The rough systolic murmur is also heard in the pulmonary area, but is loudest and roughest a little lateral from the tricuspid area. No aortic diastolic murmur is heard. There is no marked throbbing of the neck; veins in the neck are a little overfilled. There is some œdema of the ankles and overfilling of the veins of the lower extremities.

He passed over 2500 c.c. of urine daily, of specific gravity 1012–1014, containing a trace of albumen and some hyaline casts. The blood-pressure varied from 220 to 285 mm. Hg, pulse-rate 80–90.

During his stay in the hospital he had occasional smothering spells which were relieved by venesection. He spat up considerable amounts of red tenacious sputum.

The patient died during the course of the next few months. Autopsy revealed a funnel-shaped bulging of the membranous septum into the right ventricle with a perforation 3–4 mm. in diameter at the apex of the funnel. (This condition is exactly similar to the lesion described by Hebb and by Tate.)

There was also a chronic nephritis.

Case of Probable Patent Septum Ventriculorum.

B. J., an unmarried colored woman aged 26, entered the Johns Hopkins Hospital complaining of pain in the chest. Except for shortness of breath on exertion during the last ten years, the history is negative.

Her heart was very slightly enlarged to the right. The sounds were clear at the apex, but over the body of the heart a peculiar intense high-pitched murmur was heard, loudest during systole but lasting through the whole cardiac cycle (Roger's murmur?). This murmur is loudest and most intense over the third left interspace between the parasternal line and the sternum, but it is heard also in the second and fourth interspaces, where it is much less intense.

Maximal blood-pressure varied from 110 to 125 mm. Hg; venous tracings were normal; retinal vessels normal. The urine contained a trace of albumen but no casts nor blood-cells.

The subsequent history was uneventful.

DIAGNOSIS.

The diagnosis of defect in the interventricular septum can be made only when, in the absence of cyanosis or other signs of congenital heart disease, the vigorous pulsation of the right ventricle is seen in the second right interspace, and both the peculiar murmur of Roger and the accentuated second pulmonic sound can be heard. In the presence of pulmonary stenosis or other congenital or acquired lesions, signs may merge into one another in such a way that an absolute diagnosis may be impossible. Simple acquired endocarditis often occurs, as in Tebb's case, and its signs may serve further to confuse the clinical picture.

TREATMENT.

Needless to say, there is no treatment that can be directed against the defect itself. However, the symptoms in many cases arise only during over-exertion, and the most important factor in the management of the case is, therefore, directed along the usual lines for the avoidance of over-strain,—rest, graduated exercise, moderation in diet, avoidance of dyspnœa, regulation of the bowels, and if necessary digitalis and strychnine. The most important point is the avoidance of pulmonary congestion.

The prognosis in simple septal defect depends less upon the extent of the lesion than upon the apparent impairment of the function. Simple defects in the septum are compatible with quite long life, and many cases are reported in which the patients have reached the fourth and fifth decades.

When other lesions are present, such as pulmonary stenosis, abnormalities of the blood-vessels, etc., it is they, rather than the septal defect, which determines the prognosis.

PATENT FORAMEN OVALE

OCCURRENCE AND PATHOGENESIS.

Mere patency of the foramen ovale to the passage of a probe is by no means pathological. In statistics of 1166 heterogeneous autopsies collected by Vierordt, it was present 313 times (28 per cent.), and also in 80 (22.2 per cent.) of Zahn's 357 miscellaneous autopsies upon persons past the age of 40. This non-closure is probably due to the fact that both the auricular septum and the valve closing the foramen are lined by endothelium, and no fibrosis takes place between the two surfaces until the endothelial cells slough off or are injured. The valve itself remains closed against the septum during life and no symptoms are produced. Perhaps, when heart failure from any cause occurs in such cases and the pressure in the right auricle exceeds that in the left, a certain amount of blood may actually pass through this embryonic channel, but in too small amounts

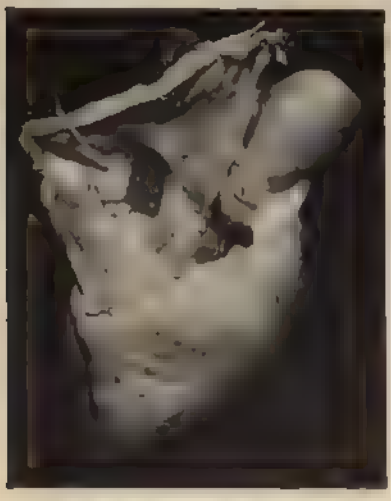


FIG. 258.—Open foramen ovale. From a specimen in the Army Medical Museum, Washington, D. C. The patient was a soldier who had never shown any cardiac signs or symptoms and no cyanosis.

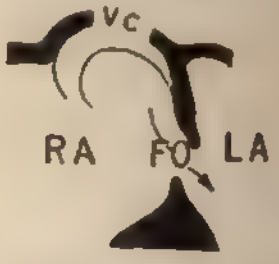


FIG. 259. Diagram showing a cross-section of the same. *VC*, vena cava; *RA*, right auricle; *FO*, foramen ovale; *LA*, left auricle.

to give signs or symptoms. On the other hand, in 462 autopsies by Hinze and by Ogle the foramen was permeable to the little finger (permanently patent) in only 9 cases (1.9 per cent.).

The mechanism of secondary septal defects (auricular stasis of Morgagni and William Hunter) by which more than the usual proportion of blood passes through the foramen ovale has been discussed under pulmonary stenosis, and it may occur with other congenital lesions.

The truly pathological lesions of the interauricular septum are shrinkage or total absence of the valve and perforation of the septum between the muscle strands (Fig. 260). Occasionally the valve has closed, but is somewhat weak and forms an aneurismal bulging. Peacock reports one and Abbott two of these cases, in all of which the protrusion was from right to left, indicating that the pressure in the right ventricle exceeded that in the left.

PATHOLOGICAL PHYSIOLOGY.

A defect in the interauricular septum has comparatively little effect upon the circulation as long as the pressures in the two auricles are equal or nearly so. When the left ventricle begins to fail or the pressure in the left auricle rises from any cause whatever (mitral stenosis, mitral insufficiency, etc.), the patent foramen ovale exercises a sort of safety-valve action and relieves the pulmonary congestion by allowing the excess of blood to pass back into the right auricle. Ritter (1856) and Rusch (1862) have shown that when the foramen ovale is open in cases of mitral insufficiency, the pulsation in the jugular vein assumes the positive ventricular type, owing to the crossing of the regurgitant stream. However, this is of little value in diagnosis, since the positive ventricular pulse is common with heart weakness and auricular paralysis.

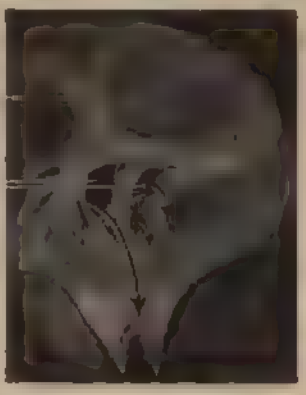


FIG. 200.—Openings between strands of muscle in the interauricular septum. From a specimen in the Army Medical Museum, Washington, D. C. The valvula foraminis ovalis (VALV. F O.) is closed and reflected at C, the rest of the septum. FOR. INTER. A. shows the openings between strands of muscle in the lower part of the septum.

On the other hand, when the right heart begins to fail and pressure in the left auricle increases, the blood follows the same course as it does in the fœtus and passes from the right into the left auricle. Under ordinary circumstances this would exert no influence whatever, and would not even produce cyanosis. But when the heart is already weak, the circulation slow, and the blood heavily charged with CO_2, this sudden admixture of venous blood carries the CO_2 content past the physiological limit, and gives rise to cyanosis and symptoms. Moreover, the blood entering the coronary arteries is also less aerated, the cardiac tonicity and cardiac strength are impaired, and the vicious circle of the open foramen ovale sets in, subsiding again with inordinate rapidity as soon as the pressure in the systemic veins falls below that in the pulmonary.

SYMPTOMS.

In most cases patency of the foramen ovale alone does not give rise to any symptoms. For example, the patient whose heart is shown in Fig. 237 was able to perform his duties as a soldier in heavy campaigns and died from dysentery without any symptoms referable to his heart. Peacock mentions the case, reported by Spry in 1805, of a girl of seven years who had no cyanosis during life and whose foramen was patent and two inches in circumference (⅔ inch in diameter). On the other hand, he cites another case, a woman of twenty-one whose foramen ovale was one inch in diameter, who from the age of three months "presented characteristic symptoms of malformation of the heart,—cyanosis, palpitation, dyspnœa, faintings, occasional convulsive attacks, and lividity."

PHYSICAL SIGNS.

Apart from the paroxysmal cyanosis the physical signs of open foramen ovale are extremely variable. Cyanosis and abnormalities in the retina may be present. On the other hand, all physical signs may be absent. In some cases systolic, in others diastolic, murmurs are present in the third left interspace at the sternal margin. Occasionally there is heard a well-defined presystolic murmur which is maximal at this point, and which when present is the most characteristic sign of the open foramen ovale.

DIAGNOSIS.

The diagnosis rests upon the presence of *paroxysmal cyanosis* and of *murmurs in the third left interspace* without signs of aortic insufficiency or of hypertrophy of the right ventricle (well-marked systolic retraction over the right ventricle with or without systolic impulse in the second left interspace) or of other congenital heart lesions. This is especially corroborated if the child was a blue baby at birth or within a few months afterwards, even if only during intervals of a few hours or days. The retinal changes are valuable signs when present.

In rare cases incidental phenomena may help in the diagnosis. Cohnheim cites a case in which the diagnosis was made from the occurrence of embolism of the brain when the primary thrombus was in the veins of the leg ("crossed embolism"); but such cases are necessarily extremely rare, and before such inferences are made all commoner factors must be carefully excluded.

TREATMENT.

Treatment between attacks of cyanosis and dyspnœa is confined to general hygiene and regulation of the patient's life, as described above for pulmonary stenosis, though, as a rule, more latitude may be allowed.

During the attacks *hot baths* and *vasodilators* (such as amyl nitrite and nitroglycerin) may be resorted to, and, when there is no diminished coagulability and the attack is severe, venesection may be performed.

PATENT DUCTUS ARTERIOSUS (BOTALLI).

PATHOGENESIS.

In many cases in which the arterial circulation is markedly disturbed in the fœtus, the ductus arteriosus (Botalli) may be found to remain patent after birth. This is a common concomitant of pulmonary stenosis and especially pulmonary atresia, of the corresponding conditions at the aortic orifice, and of congenital lesions at either of the auriculoventricular valves.

When viewed in the light of its closure, the mechanism of this secondary non-closure of the ductus is tolerably clear. The ductus arteriosus Botalli represents the remains of the sixth branchial arch (Fig. 240). It "is in a direct line with the pulmonary trunk, is the direct continuation of the same, and is of almost equal size, while it is of greater diameter than the descending arch of the aorta. A distinct narrowing of the aortic arch is to be observed just above the entrance of the ductus into it." (Klotz.)

Closure of the Ductus Arteriosus. Several theories have been advanced to explain the closure of the ductus arteriosus at birth:

Haller thought that it results from coagulation of the blood within its lumen. Kilian (1828) was the first to show that with the expansion of the lungs at birth the resistance in the pulmonary circulation was diminished and a large amount of blood thus diverted from the channel through the ductus. A number of theories have been proposed to explain the exact manner in which this diversion of blood through the pulmonary channels brings about the closure of the ductus arteriosus.

Strassman (1894) attempted to explain the closure on purely mechanical grounds. He called attention to the fact that the ductus arteriosus penetrates the wall of the aorta at an acute angle, so that the tissue included in this angle forms a sort of valve. He believed that when the pressure within the aorta became greater than that in the pulmonary artery (after birth), this flap of vessel wall closed down over the mouth of the ductus and prevented blood from entering it. Strassman found moreover that if he injected fluid into the aorta of a new-born child at a pressure under 100 mm. no blood entered the pulmonary artery. These experiments have been very carefully repeated in a large number of infants by Klotz, who found that "at all times when the ductus arteriosus was unobliterated by new-formed or forming fibrous tissue the colored fluid found its way into it for some distance sufficient to stain it." However, the fact that the communication was not a free one demonstrates that this valvular action is probably a contributing cause in cutting off the blood flow or in lessening the pressure in the ductus.

On the other hand, Schulze in 1871 showed that the walls of the ductus arteriosus, though poor in or lacking elastic fibres, were particularly rich in muscle fibres. He believed that when the blood-pressure in the pulmonary artery fell, and the blood was diverted away from the ductus arteriosus, the muscle fibres in the wall contracted down further until the lumen was finally obliterated. This occurs without any such intravascular coagulation as Haller had supposed, but Langer (1857) has found that it is accompanied by a very active proliferation of the cells in the intima, with sloughing off of the endothelial lining (Klotz). The proliferation goes on till the wall of the vessel becomes thicker than that of either the pulmonary artery or the aorta, and it is finally occluded by fibrosis.

Factors Causing Persistence of Ductus. Under pathological conditions it is clear that anything which causes obstruction to the flow of blood through the arch of the aorta during fetal life (aortic stenosis or atresia, congenital mitral stenosis, coaretation of the arch of the aorta, etc.) will cause the right ventricle to carry on the greater part of the circulation and to force more blood than usual through the ductus arteriosus. This condition, of course, persists after birth; the ductus, which now represents a main blood channel, remains open. The flow continues in the usual direction backward from the pulmonary artery into the aorta.

On the other hand, when there is atresia of the pulmonary artery the pressure in the ductus is low and blood enters it from the aorta, passing f o w a r d (ventrally) into the rami pulmonales. These facts explain the persistence of the ductus arteriosus in its usual occurrence as a secondary congenital lesion.

The occurrence as a primary lesion is rare, only 26 cases having been collected by Vierordt in 1898, 12 more by Abbott. Klotz believes that these may be "the result of imperfect expansion of the lungs. In these cases the blood-pressure has never been lowered in the pulmonary system to the point which allowed the walls of the ductus to overcome it." It may also result from congenital weakness of the left ventricle causing a low blood-pressure in the aorta at the time of birth.

The size and structure of the patent ductus may vary greatly, from a short and narrow passage to an almost aneurismal dilatation. Acute or

malignant vegetations are not uncommon within the lumen, and the presence of an open ductus tends to predispose to endocarditis. Arteriosclerosis of both the ductus and the pulmonary artery also occurs, perhaps as a result of the high pulmonary pressure.

SYMPTOMS.

The symptoms of uncomplicated patent ductus arteriosus are usually obscure and slight, and the condition is often found incidentally. Cyanosis is slight and transitory. Slight weakness and shortness of breath on exertion may occur, but many of the cases are devoid of symptoms.

PHYSICAL SIGNS.

Several diagnostic features of open ductus arteriosus have been published at various times. Gerhardt in 1867 described a small quadrilateral extension of the area of dulness in the second (and first) left interspace. In this region the pulsation of the pulmonary artery may be seen, and the well-marked systolic retraction is often seen over the interspaces corresponding to the hypertrophied right ventricle. Zinn, de la Camp, and others have found, on examination with the fluoroscope, that Gerhardt's area of dulness corresponds to a round shadow of a small mass along the left upper margin of the cardiac shadow, where the pulmonary artery and left auricle are usually seen (Fig. 261). This mass shows systolic pulsation and corresponds to the dilated ductus arteriosus. This dilatation is frequently aneurismal. The picture with the X-ray is thus of great diagnostic value, but one must carefully exclude an aneurism of the aorta behind the sinus of Valsalva. In contrast to the shadow of the left auricle, this shadow is magnified when the tube is placed in front of the body as compared with the illumination from behind.

The signs on auscultation vary considerably. There is usually a systolic murmur over the precordium, and especially over the second left interspace, which sometimes replaces but often follows the first sound. This murmur when not caused by another concomitant lesion is due to the rush of blood through a narrowed ductus into the wider lumen of the aorta. François-Franck has shown that this murmur is heard loudly at the left back over the area at which the aorta comes in contact with the chest wall (level of the third and fourth spines), to which it is transmitted in a direct line (Fig. 256). He showed that the loudness of this murmur bears a definite relation to the phases of respiration. It is loudest during expiration, for at that time the resistance in the pulmonary circuit is greatest, and hence the blood flows through the ductus more rapidly, but becomes feebler in inspiration when more blood passes through the lungs and less through the ductus. François-Franck also found that this variation in the blood flow into the aorta found equally marked expression in the pulse, causing a rise of pressure and full pulse in expiration, fall and small pulse during inspiration (pulsus paradoxus).

These signs, however, occur only in cases in which there is an efficient circulation through the pulmonary orifice. In the cases associated with

pulmonary atresia where the flow through the ductus is in the opposite direction, the murmur may be absent at the back and will be loudest during inspiration, for then the inflow into the lungs is greatest. Nevertheless, the pulse will remain a pulsus paradoxus, for the flow through the aorta during inspiration will be diminished just the same. This respiratory variation of murmur and pulse is, however, often absent in spite of the open ductus, as is the systolic murmur itself in some cases. Neither bears an absolute relation to the degree of patency of the ductus.

FIG. 261.—Radiograph of a thirteen-year-old boy with patent ductus arteriosus (Botalli) and aneurismal dilatation of the ductus and pulmonary artery. (After Hochsinger, of Pfaundler and Schlossmann's "Diseases of Children.") *AA* arch of the aorta; *DB* ductus Botalli and pulmonary artery dilated, the ductus aneurism giving a cup-shaped top to the shadow of the heart; *MI* internal mammary artery, considerably dilated, denoting an internal collateral circulation.

A diastolic murmur is often heard along with the systolic, sometimes replacing the second sound but more often accompanying or following an accentuated pulmonic sound. The inequality in pressure between aorta and pulmonary artery persists during diastole, and the abnormal blood flow therefore continues and produces the murmur in diastole. When the difference of pressure is slight, especially with low peripheral resistance, the diastolic murmur may be absent.

DIAGNOSIS.

From the above discussion the points upon which the diagnosis of the open ductus arteriosus may rest are sufficiently clear,—pulsation over the right ventricle, Gerhardt's dulness, a systolic or double murmur loudest at second left interspace and heard at the left upper back, expiratory accentuation and pulsus paradoxus, and the pulsating mass in Gerhardt's area seen on X-ray examination. In addition, the history may show that the patient was blue at birth (before the pulmonary channels have opened up) but that cyanosis soon passed off.

J. Plesch in Kraus's clinic (Berl. klin Wchnschr., 1909, xlvi, 391) has attempted to make the diagnosis by analyses of the expired air. By a very simple device he determines the percentage to which the blood flowing through the pulmonary artery is saturated with oxygen. Under normal conditions the saturation is 38-70 per cent. of its oxygen capacity. In patent ductus arteriosus the blood in the pulmonary artery is mixed blood and hence its oxygen content is higher (80-90 per cent.). The aerated blood could enter only through a patent ductus arteriosus.

TREATMENT.

Treatment for the persistence of the ductus consists mainly in those methods which improve pulmonary circulation,—breathing exercises, careful hygiene, avoidance of exposure to pulmonary infections, and avoidance of fatigue, general muscular and cardiac overstrain. Since the persistence of the ductus is in itself a compensatory process, it calls for no special remedy. To ligate it, as might readily be done after opening up the thorax under positive pressure, would be harmful rather than beneficial. Otherwise general hygienic measures and cardiac stimulants are of value, as in other diseases. But in many cases open ductus Botalli has no effect upon the duration of life and requires no treatment.

STENOSIS OF THE AORTA

Stenosis of the lumen of the aorta may occur in three places:

I. At the aortic valve.

II. Stenosis of the arch of the aorta.

1. Above the entrance of the ductus arteriosus Botalli.
2. Just below the entrance of the ductus arteriosus Botalli.

I. Stenosis at the aortic orifice is one of the rarer congenital lesions (2 per cent. of Abbott's series), though probably many of the milder cases escape detection. It is usually due to endocarditis late in fetal life.

Those which develop earlier in fetal existence, in which true aortic atresia occurs, are quite analogous to the cases of pulmonary atresia, except that the posterior instead of the anterior channel of the common truncus arteriosus fails to develop. The changes in the fetal circulation are similar to those in pulmonary atresia, but affect the opposite sides of the heart. The septa remain open, and occasionally one ventricle (the left) fails to develop. Practically the entire systemic circulation is carried on by the pulmonary artery through the ductus arteriosus.

The consequences of the lesion are very severe and few cases survive birth, in striking contrast to pulmonary stenosis and atresia. No doubt

this is due to the fact that, since the right ventricle is the stronger in fetal life, it succeeds in establishing a better compensatory circulation after atresia of its orifice than does the left. Moreover, when the first breath is taken after stenosis of the aorta, it is venous instead of arterial blood which is thrown into the organs.

II Stenosis in the vicinity of the ductus Botalli is one of the most common congenital heart lesions, occurring in 198 of Abbott's 412 cases. Like most abnormalities it arises as an exaggeration of a condition which is normally present in the fœtus. As stated above, Klotz finds that there is a distinct narrowing of the aortic arch just above the entrance of the ductus into it. This is no doubt due to the fall of pressure in the aorta which occurs below the left carotid artery and the rise further on when the blood enters from the ductus.

Hamernik in 1844 divided the cases into: (1) stenosis above the ductus, (2) those at the entrance of the ductus, and (3) those below the ductus. Bonnet, who made an exhaustive study of the subject in 1903, discards Hamernik's second group, and distinguishes two types:

1. The type in the new-born, in which the stenosis occurs above the ductus and the latter remains open.
2. The type found in adults, in which the stenosis occurs below the ductus. The latter is closed and collateral circulation develops.

Bonnet's studies were based upon 160 cases, of which 55 (34.3 per cent.) were of the new-born type and 105 (65.7 per cent.) were of the adult type.

Type of the New-born.—The cases of the new-born type represent an exaggeration of the slight narrowing in the aorta which, as Klotz states, is present above the left subclavian artery and the ductus Botalli. Embryologically, as Longa points out, this represents the branch joining the fourth and sixth branchial arteries (Fig. 240) and might correspond to a failure of development of this branch. On the other hand, the amount of blood in the aorta is very much depleted by the flow into the innominate, left carotid, and subclavian, so that its lumen is naturally smaller until replenished by the inflow from the ductus. There is consequently a region of functional stenosis between these two points which may be exaggerated by contraction of the muscle-fibres in the wall of the aorta. The weaker the action of the left ventricle the more marked will be this functional stenosis. The ductus Botalli therefore takes on more and more of the circulation in the lower parts of the body, and fetal life may be undisturbed as long as the right ventricle is pumping aerated blood; but when this condition ceases and the pulmonary channels widen and pressure in the ductus falls, the aortic circulation may become insufficient and the syndrome of

Fig. 262.—Stenosis of the isthmus of the aorta above the ductus arteriosus. Infantile type of the new-born. (From a specimen in the Army Medical Museum, Washington, D. C.)

congenital heart disease may result. It is rare for these children to live more than a few weeks, or at most a few months, and many die at birth.

Physical signs are indefinite, confined to double murmurs over the chest and back and in most cases cyanosis. There are very often associated malformations, such as pulmonary stenosis, open septum ventriculorum, etc.

In this form there is very little attempt at establishment of a collateral circulation, since the greater part of the systemic circulation is maintained by the right ventricle through the open ductus arteriosus, just as it is before birth. Owing to the completeness of this compensation, there may be little difference between the pulses in the upper and in the lower extremities, and the clinical diagnosis is scarcely ever made

Adult Type. — In the second or adult type, which is more common, the stenosis occurs just below the entrance of the ductus Botalli, and this vessel is found to be closed. Indeed, the very stasis at this point assists in its closure. The mode of origin of the stenosis at this point is not clear Bonnet calls attention to the fact that the lumen of the aorta at the stenosis (usually 2 4 mm.) is about that of the normal aorta at the time of birth, and thinks that the whole anomaly may be of postnatal development It is possible, as he suggests, that, when the ductus Botalli is particularly long, the fibrosis of the latter brings about a kinking of the aorta at this point, and with a dilatation above and stenosis at the point of kinking.

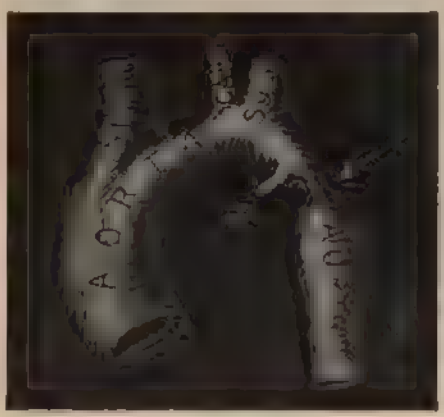


FIG. 263.—Stenosis below the ductus arteriosus Botalli adult type After Bonnet. Rev. de Méd., Par 1903 xxiii

Skoda, on the other hand, has suggested that the stenosis may result from a band of fibrosis passing around the aorta at this point: but, though this theory is alluring, there is no definite histological evidence in its support.

The stenosis cuts off the circulation from all parts of the body below the stenosis, but the high pressure due to the stagnation above it causes a progressive dilatation of other arterial channels, such as the mammaries, thoracic and scapular arteries, which are always found to be much dilated. Indeed, the collateral circulation may be so good that the lumen of the aorta below the stenosis may be as great as above it (hour-glass constriction), though usually it is somewhat narrow and it may even be funnel-shaped

Clinically the presence of this type of stenosis does not necessarily shorten life, though this depends largely upon the completeness of the collateral circulation. The symptoms are chiefly those of cerebral congestion, headache, vertigo, buzzing in the ears Occasionally there are pains in the chest. Bonnet calls attention to the fact that in his 105 cases there was never intermittent claudication, showing that the circulation in the lower limbs is always sufficient.

PHYSICAL SIGNS AND DIAGNOSIS.

Cyanosis is not common and not a sign of the disease. The most definite indication is the difference in the size and quality of the pulse in the upper and lower extremities, the carotid and radial pulses being large and throbbing, the femoral, popliteal, and dorsalis pedis as well as the abdominal aorta small or impalpable. Though the diagnosis *intra vitam* is rare, Lepine was able to make it from these data in two cases. Dr. W. S. Morrow calls attention to the possibility of diagnosis from marked difference in the brachial and tibial blood-pressures, but just as in aneurisms the difference in size and quality of the pulse on palpation would usually be more marked than that of the blood-pressures. Moreover, Halsted has found little difference between brachial and femoral pressures in man after the abdominal aorta has been occluded with metal bands for the treatment of aneurism. The presence of large tortuous mammary, thoracic, and scapular arteries aids in the diagnosis. There are usually low murmurs over the arteries, especially at the angle of the left scapula, as was present in a case diagnosed by Mercier in 1839.

Valvular disease of the heart frequently results from the increased work thrown upon the heart and dilatation of aortic and mitral orifices, and their signs complicate the picture. Before making the diagnosis, it is always necessary to exclude aneurism and mediastinal tumor by the absence of dulness on percussion and of abnormal shadows on X-ray examination.

TREATMENT.

Treatment depends purely upon symptoms, occasional venesection being of value to relieve the headaches. It is most important for the patient who suffers from these symptoms to avoid over-exertion or excitement, which cause too vigorous action of the heart.

DIFFUSE NARROWING OF THE AORTA.

Virchow has also called attention to another form of abnormality in the lumen of the aorta, a diffuse narrowing of its entire lumen throughout its whole extent (hypoplasia of the aorta). This condition is associated with under-development of the elastic and muscular elements in the arterial walls. As Virchow and other observers have found, it is often associated with chlorosis of intense grades and occasionally accompanies other congenital malformations of the heart.

About the objective finding there is little dispute. The only point in question is whether the condition is to be regarded as a true congenital malformation or as a postnatal development, which, like the changes in rickets, is determined by conditions of growth and nutrition during childhood and may be corrected by cure of these conditions. It is possible that it may be secondary to the conditions which bring on anæmia, and due to the fact that the aortic walls have never been subjected to the stimulating influence of an adequately high blood-pressure. That this may be a factor in the development of and strengthening of blood-vessel walls has been shown especially by the results of arteriovenous anastomosis and trans-

plantation, in which the walls of the transplanted vein become thicker and richer in elastic and muscular elements (Carrel). It is of course extremely difficult to determine what would have occurred if such cases had recovered from their anæmia or primary debility and blood-pressure had reached a normal level. It is equally difficult to determine that any such cases have recovered under these conditions, though the fact that the lumen of the radial artery increases (pulse becomes larger) with the recovery from chlorosis is of course definite.

For the present, therefore, one must hesitate somewhat in classing hypoplasia of the aorta among the definite congenital malformations.

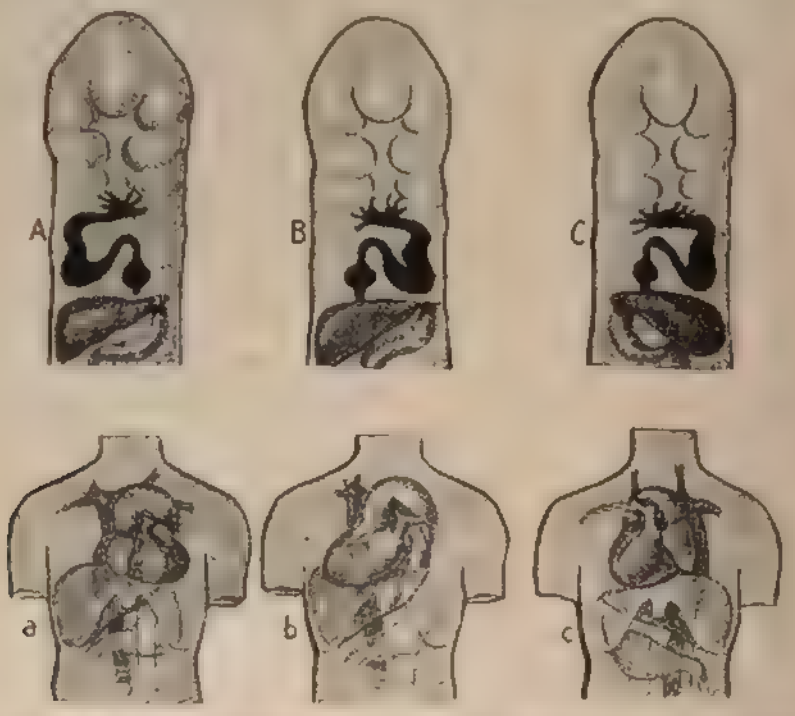


Fig. 264.—Transposition of the viscera in embryo and adult. Schematic. *A B C*, position of organs in the embryo; *a b c*, position of organs in the adult. *A a*, normal; *B b*, transposition of the heart and arteries, simple dextrocardia; *C c*, complete situs transversus.

COMPLETE AND PARTIAL SITUS TRANSVERSUS

It is not extremely rare to meet with a case of complete transposition of the viscera, so that the heart and stomach are found to lie on the right side (dextrocardia, dexiocardia) and the liver upon the left. This condition is probably brought about by a change in position of the cardiac tube in early embryonic development, so that it lies in the position of ƨ instead of the normal S (Fig. 264). Maude Abbott suggests that in these cases there is a change, the embryo lies in an abnormal position within the chorion so that its right side instead of its left is closer to the blood supply. At all events the relation of the organs is the mirror image

of the normal condition. In complete transposition, however, the organs develop normally, and the condition, though unusual, has no effect upon the function. Persons whose hearts lie on the right side are quite as free from symptoms as those whose hearts are on the left, provided the other viscera are normal; and the condition is usually discovered accidentally during routine physical examination. In such cases the apex impulse and

FIG. 205. Transposition of the valves. From a specimen in the Army Medical Museum, Washington, D. C. The course of the interventricular septum is indicated upon the heart wall. The course of the blood current is shown by the arrows.

heart sounds are heard in the fifth right interspace, and the second aortic is heard on the left side instead of on the right. In persons with thick or barrel-shaped chests, and especially in women whose breasts are large, the condition is readily overlooked.

Dextrocardia (dexiocardia) without transposition of other viscera is much rarer. Most frequently it is due to a pushing or pulling of the heart to the right by intrathoracic growths or adhesions, but occasionally (in 2

FIG. 206. Pulmonary artery with four cusps. From a specimen in the Army Medical Museum, Washington, D. C.

of Abbott's 112 cases) it is due to alteration in development. Under these circumstances the pulmonary artery is given off from the left ventricle, the aorta from the right. Great variations may be seen in the arrangement of venæ cavæ, which sometimes enter the left, sometimes the right auricle. The results, as in other cases in which the blood is mixed, vary greatly. The syndrome of congenital heart disease may be present, owing to the mixing of blood, but the exact transposition of vessels can rarely be diagnosed *intra vitam*.

ABNORMALITY OF THE VALVES

The number and formation of the cardiac valves may also undergo alteration in fetal life. In the aortic and pulmonic this is usually due to inflammatory fusion of two cusps forming a bicuspid valve (Fig. 265), or to the fact that one of the leaflets is divided into two parts by a slit and finally under the influence of the blood-pressure grows to form symmetrical cusps (Fig. 266).

In the mitral and tricuspid valves, especially in association with open septum ventriculorum, there may be a split in the middle of one leaflet,' practically converting it into two separate cusps. Except for the formation of valvular insufficiencies which result, multiplicity or paucity of the cusps has no pathological effects.

A large number of other malformations, such as partial separation of the two ventricles to form a "bifid apex," defective formation of the chest wall with exposure of the heart (ectopia cordis), malposition of the heart causing it to lie in the abdomen or the neck, transposition and malformation of the great arteries and veins, are encountered. Space does not permit of a complete discussion of these conditions, for which the reader is referred to the magnificent article by Dr. Maude Abbott in Volume IV of Osler's Modern Medicine.

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XI.

HEART-BLOCK AND THE ADAMS-STOKES SYNDROME.

HISTORICAL.

In 1827 Robert Adams, of Dublin, reported the case of a revenue officer, aged 68, whose pulse-rate was 30 per minute and who suffered from dyspnœa, cough, and attacks of fainting ("apoplectic attacks"), "during which his pulse would become even slower than usual. . . . He recovered from them without paralysis." In the same year an exactly similar case was reported in great detail by Wm. Burnett. Burnett's observations were reported even more carefully and in greater detail than those of Adams and ill deserve to have fallen into oblivion. Burnett further called attention to the fact that Morgagni had described two cases of "epilepsy with slow pulse" in 1761. Holberton described another case in 1841, but general attention was not attracted to the condition until Wm. Stokes published four cases in 1846.

Since then the condition of *persistent extreme bradycardia with syncopal or convulsive seizures has been known as the Adams-Stokes syndrome*, though it may more accurately be designated by the names of Morgagni-Adams-Stokes, as Pletnew has done, or by that of Morgagni-Adams-Burnett or Adams-Burnett syndrome.

As but little can be added to the clinical descriptions of these cases, one of Stokes's histories may be reported in some detail:

"Edmund Butler, aged 68, stated that his health had been robust until three years before admission, at which time he was suddenly seized with a fainting fit. This occurred several times during the day and always left him without any unpleasant effects. Since that time he has never been free from attacks for any considerable length of time, and has had at least fifty such seizures. The fits are very uncertain as to their period of invasion and very irregular as to their intensity, some being much milder and of shorter duration than others. They are induced by any circumstance tending to impede or oppress the heart's action, such as sudden exertion, distended stomach, or constipated bowels. There is little warning given of the approaching attack. He feels, he says, a lump first in the stomach, which passes up through the right side of the neck, where it seems to explode and pass away with a noise like thunder by which he is startled. This is often accompanied by a fluttering sensation about the heart. . . . At first he found that spirits were the best restorative or prophylactic, but latterly he has not used them, being 'afraid to die with spirits in his belly.'

"On admission he was haggard and emaciated. . . . On percussion the chest is universally resonant. The respiratory murmur is louder and combined, especially posteriorly, with large mucous rales. The impulse of the heart is slow and of a dull heaving character. . . . The first sound is accompanied by a soft *bruit de soufflet*. The second sound is also imperfect. . . . We remarked to-day that on listening attentively to the heart's action we perceived that there were occasional semi-beats between the regular contractions — very weak, unattended with impulse, and corresponding to a similar state of the pulse, which thus amounts to about 28 in the minute, the evident beats being only 28. . . .

"(June) The cardiac phenomena remain as before, but a new symptom has appeared, namely, a very remarkable pulsation in the right jugular vein. This is most evident when the patient is lying down. The number of reflex pulsations is difficult to be established, but they are more than double the number of manifest contractions. About every third pulsation is very strong and sudden and may be seen at a distance, the remaining waves are much less distinct.

"He has scarcely had any of the cardiac attacks since he was discharged."

THE CONCEPTION OF HEART-BLOCK.

Stokes did not appear to have any definite understanding of the nature of these "semi-beats" nor of the functional disturbance associated with them. A similar, more accurate observation was made by A. Chauveau. Chauveau in 1882 made observations upon a case whose usual pulse-rate was 24 per minute, and who suffered from occasional attacks of vertigo and loss of consciousness. Tracings made from the apex showed a series of large beats at regular intervals corresponding to the loud heart sounds and to the radial pulse, and also a second series of very small notches occurring at equally regular intervals but bearing no relation whatever to the beats of the ventricle. As in Stokes's case, these small pulsations were accompanied by "small sounds which may give the illusion of reduplication of either heart sound." The usual presystolic notch due to the auricular beat was absent, and Chauveau correctly concluded that these small notches were due to the contractions of the auricles, which were beating at a rate of 66 per minute while the rate of the ventricles was 24. Chauveau investigated the matter experimentally, and was able to demonstrate that in horses upon stimulation of the vagus the auricles could be observed to beat more frequently than the ventricles. He therefore naturally considered the dissociation of auricular and ventricular rhythm as due to over-stimulation of the vagus. This conclusion was further warranted by the fact that his patient had pains at the back of the neck, and the chief of service had diagnosticated a lesion of the medulla at the level of the vagus nucleus.

FIG. 267.—Tracing of the apex beat in a case of Adams-Stokes disease. After Chauveau.

While Chauveau was experimenting in France, experimenters in Germany and in England were unconsciously throwing light upon the condition from different stand-points. In 1883 Wooldridge, under Ludwig's direction, was investigating the course of the nerves in the cardiac septum, and for this purpose constricted the interauricular septum by tightening a fine silk ligature introduced so as to embrace only the septum. He observed "the auricles and ventricles continue to contract, but no longer with equal frequency. Stimulation of the vagus causes the auricles to stop beating, the ventricles continue.

"The ligature is removed, at first the auricles and ventricles beat at different rhythms, then the uniform (normal) beats of both chambers return, and stimulation of the vagus now inhibits both auricle and ventricle."

Wooldridge's results were confirmed in 1884 by Tigerstedt, who cut through the septum with a specially devised "atriotome."

These observations under Ludwig's direction were made with the view only of cutting the intracardiac nerves. As a matter of fact, the muscular connections were severed as well, but the importance of these was disregarded.

The myogenic conduction from auricle to ventricle was, however, at this very period being demonstrated by Gaskell in Cambridge upon the heart of the tortoise and frog, in which the auriculoventricular function is represented by a wide band of muscle whose properties differ somewhat from those of either the auricle or the ventricle. Gaskell demonstrated that "if this auriculoventricular ring were clamped, the auricle continued to beat at unaltered rhythm, but as the clamp was tightened the period between auricular and ventricular contractions (A_s–V_s interval, on conduction

FIG. 208.—Partial heart-block. 3 : 1 rhythm produced by pressure upon the vagus in a patient with disturbed conductivity who was also subject to attacks of the Adams-Stokes syndrome. Tracing made by Dr. J. W. Peabody and the writer. 4, 4, 4, 4, auricular contractions to which the ventricles do not respond.

time) was gradually lengthened; then the ventricle failed to respond to some of the impulses from the auricle, and, according to the tightness of the clamp, the ventricle could be made to . . . respond to every second contraction of the auricles (partial heart-block), to respond to every third, fourth, or other contraction, or to remain quiescent. When the clamp was closed very tightly the ventricle remained still for a variable time, then, in accordance to its inherent rhythmical power, developed a rhythm of its own (rhythm of development), the rate of that rhythm when fully developed and the length of time that the standstill lasted being correlated with the rhythmicity of the tissues." The condition in which the ventricular ventricle no longer follows any of the impulses from the auricles is termed complete heart-block, in contrast to the partial heart-blocks in which the impulse ventricle is responding to some, but not all, of the impulses arising in the auricles.

Gaskell showed that heart-block also set in when the bridge of tissue connecting the auricles and ventricles was cut down to a sufficiently narrow strip. He was able to produce similar blocks between portions of the

auricle or ventricle by clamping or cutting, just as Romanes had done for the muscle in the bell of the medusa. Gaskell demonstrated also that the block between auricles and ventricles remained complete when the only connection between the auricles and the ventricles was formed by the coronary nerve.

THE AURICULO(ATRIO)VENTRICULAR MUSCLE BUNDLE.

Anatomy.—The existence of muscular connections between the auricles and ventricles in man and mammals was, however, denied until 1893, when Stanley Kent, of Oxford, found that in the rat and other mammals there was a large strand of small fusiform muscle cells with fusiform nuclei which ran in the septum membranaceum and connected the musculature of the ventricle with that of the auricle.

In the same year Wm. His, Jr., described the presence in the mouse, dog, and man of a bundle of muscle-fibres which "arises from the posterior wall of the right auricle near the inter-auricular septum, in the atrioventricular groove, lies upon the upper edge of the muscular interventricular septum, passes forwards and to the vicinity of the aorta, where it divides into a right and a left branch. The latter passes down to the base of the anterior mitral cusp."

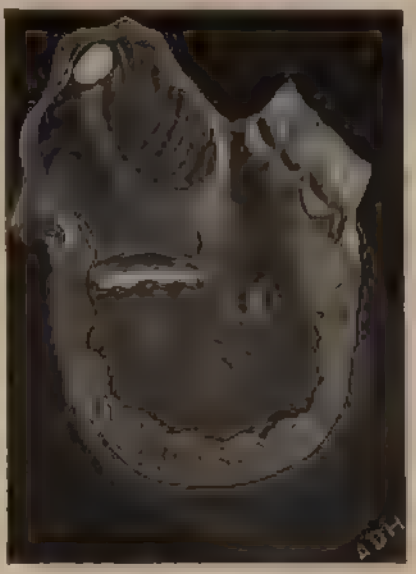

Fig. 269.—The right branch of the auriculo-ventricular bundle in [illegible]'s heart. (After Barker and Hirschfelder, Arch. Int. Med., 1909.)

These anatomical findings of His have been confirmed by Braeuing, Humblet, Retzer, and Tawara. The latter found that the fusiform cells described by Kent were really Purkinje fibres, and that the muscle bundle of His is in reality continuous with the entire network system of Purkinje fibres which permeates both ventricles. Tawara also demonstrated the presence of nerve-fibres within the His bundle, and Gordon Wilson has recently demonstrated ganglion cells as well.

In a later research Retzer has stated that this conducting system is continuous above with the septal portion of the right auricle (Fig. 269), and that its cells are of the same histological structure as those about the sinus region. He believes that it is a true sinoventricular bundle, but the recent work of Lydia de Witt seems to confirm the claims of Tawara. The idea that the cardiac impulse must pass from sinus to auricle before reaching the auriculoventricular bundle is borne out also by the observation of Dr. G. S. Bond that in the frog the auriculoventricular muscle can be seen to contract considerably later than the auricle but before the ventricle.

Experimental Physiology of the Auriculoventricular Bundle.—The first experiments upon the physiology of heart-block in mammals were

performed by Stanley Kent, the discoverer of the auriculoventricular bundle, in 1893, and were recorded by him in his original publication in the following words: "By the use of a suitably constructed clamp . . . I have been able to verify for the mammal (*i.e.*, in the excised heart of the rat), almost all the effects described by Gaskell as obtained in the frog." Kent thus seems to have forestalled all the later experiments upon the subject, but the brevity of his physiological note left much to be investigated. In 1895 His repeated the experiments of Wooldridge and Tigerstedt, and demonstrated that in order to bring about dissociation of the auricles and ventricles it was not necessary to injure the entire septum but merely this auriculoventricular muscle bundle.

In 1899 he applied his anatomical and physiological studies to a case of Adams-Stokes disease, in which he confirmed Chauveau by finding independent action of the auricles and ventricles, and designated this by Gaskell's term "heart-block." His also gave an excellent tracing, taken during a syncopal attack, demonstrating that the auricles continued at their usual rate while the ventricles ceased to beat for several seconds and then resumed their beat at a gradually increasing rate (corresponding to Gaskell's "rhythm of development").

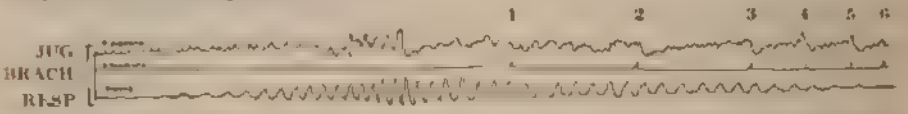

FIG. 270. Tracings from the carotid artery and jugular vein of a patient with Adams-Stokes disease, showing stoppage of the ventricles and continuance of the auricular contractions during the attack. After His *Deutsches Arch. f. klin. Med.*, 1899, lxiv.) 1, 2, 3, 4, 5, 6 represent the onset of independent ventricular contractions.

The experiments of Kent and His and their predecessors were confirmed by Humblet, Hering, and Tawara, and led to the conclusion:

1. That the slow pulse of Adams-Stokes disease was due to dissociation between the auricles and ventricles (heart-block) and to the slow independent rhythm of the ventricles.

2. That the syncopal attacks (Adams-Stokes syndrome) were due to cessation of ventricular beat but not of the auricular beat.

Experimentally they had produced the former but not the latter in mammals, while Gaskell had produced it in the frog and tortoise. Nor had pathological changes in the auricular ventricular bundle been shown in cases dying from the Adams-Stokes syndrome. The missing link was supplied by American scientists.

Factors Affecting Degree of Heart-block. — In 1904 Erlanger began a series of experimental and clinical investigations upon this condition. He first confirmed all of His's findings in man, and refuted Chauveau's claim, that the heart-block was due to the vagi, by showing that when in his cases the latter were paralyzed with atropine the heart-block did not pass off.[1] Then he devised a modification of Gaskell's clamp, an L-shaped hook of steel wire whose arm could be pressed against a brass block by means of a bolt and screw.

[1] Edes had previously shown that belladonna had no effect in his cases of Adams-Stokes disease.

The hook was introduced into the right wall of the aorta just above its origin (the pericardial fat having been dissected off), the point passed backwards and downwards into the left ventricle, and then pushed through the ventricular septum till it entered the right ventricle (Fig. 271). The brass block was then pushed down over the long arm of the L and the nut gradually screwed taut. The first effect observed was lengthening of the conduction time (A, V, or A V interval): then alternate ventricular beats disappeared (2 : 1 rhythm), at first occasionally, then regularly. With further tightening of the clamp a 3 : 1 rhythm occurred, and finally complete heart-block. "After the ventricles have emptied themselves it may be seen that each contraction of the auricles sends into the former a distinct wave, upon the subsidence of which the volume of the ventricles is seen to have been considerably increased." In many but not in all of his experiments the complete block began with a complete stoppage of the ventricles, exactly like that in the Adams-Stokes syndrome, in which "the ventricles stop beating without warning. The auricles continue to beat with an apparently unaltered rate . . . . The ventricles enlarge with each contraction until their distention becomes really huge . . . . Respiratory convulsions may begin. Witnesses are almost convinced that the experiment has come to a close when it may be that after more than twenty seconds the ventricles suddenly empty themselves with one great effort." This is sooner or later followed by another and another until the slow ventricular rate is gradually assumed.

Fig. 271.—The Erlanger heart-block clamp compressing the auriculoventricular bundle (A V B). S M, septum membranaceum, M V, mitral valve.

Factors Affecting Stoppage of the Ventricles.—This "stoppage" of the ventricles represents the condition which is the cause of death and discomfort in man. Patients, as a rule, remain free from symptoms while the pulse-rate remains regular. A study of the factors bringing it about was therefore of the utmost importance. Erlanger was able to show that it was neither brought on nor prevented by stimulation of the cardiac nerves when the heart was in a condition of either partial or complete heart-block; indeed the cardiac nerves exert less effect than upon the

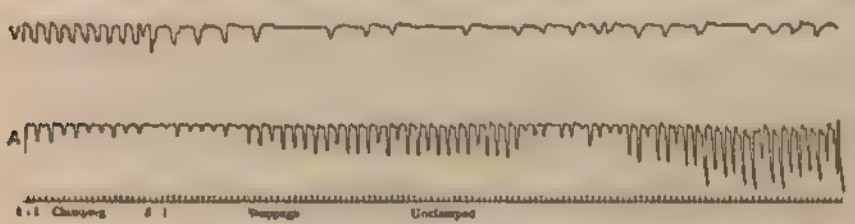

Fig. 272.—Effect of gradually tightening the clamp. (After Erlanger and Hirschfelder.) Shows 2 : 1 rhythm, finally stoppage of the ventricles with complete block. After this the ventricles can be seen to contract at an independent rhythm.

uninjured heart. Erlanger and Hirschfelder investigated the subject still further, and found that stoppage of the ventricles occurred when the clamp was tightened rapidly and a complete block produced suddenly. It occurred more rarely when the clamp was tightened slowly, and the heart was allowed to pass through the various stages of partial block (2 : 1 and 3 : 1 rhythm). In only two experiments did it occur after the ventricles had already taken on their independent rhythm. Whenever from any cause (stimulation of the accelerators, application of heat, or rhythmic induction

shocks) the rate of the auricles was increased, the degree of block was also increased, a normal rhythm (1 : 1) passing to a 2 . 1, a 2 : 1 rhythm to a 4 : 1 or to complete block. When this occurred rapidly stoppage of the ventricles sometimes set in. Conversely, slowing of the auricles from any cause (stimulation of the vagus, application of cold, etc.) improved conductivity and facilitated the passing off of the block.

In this respect the experimental heart-block differs greatly from the clinical, since in a number of cases (Gibson, Thayer) it has been found that stimulation of the vagus increases the degree of block while atropine removes a partial but not a complete block. In other cases (Edes, Erlanger, Schmoll) it has no such effect.

The duration of the period of "stoppage" (during which the ventricles remained quiescent) varied greatly, and was greatest in those hearts which could be inhibited longest by stimulation of the vagus. As in Gaskell's tortoise, it seemed to be definitely "correlated to the rhythmicity" of the ventricles, which is greater in some hearts and at some stages of the experiment than at others. In general it has appeared to the writer that the poorer the condition of the ventricular muscle the longer the period of stoppage. Slight asphyxia, though it did not in itself bring about stoppage of the ventricles, seemed to lengthen the period of stoppage from clamping.

In some experiments the ventricle remained quiescent for so long (more than 55 seconds) that the animal would have died at once had not the heart been revived by mechanical stimulation.

In a subsequent paper Erlanger has shown that the condition of block on clamping or injury depends upon the condition of the cells in the His bundle. Each cardiac impulse leaves them in a condition of lowered irritability from which they recover gradually. When the injury is slight they recover just too late for the next impulse from the auricle and are only ready to receive the second stimulus (a 2 : 1 rhythm resulting). When they are injured a little more they recover in time for every third or every fourth impulse, and finally the stimulus always remains below the threshold of irritability and complete block sets in. Similarly, the more rapid the rhythm the less time the cells have had to recover and the less the intensity of impulse from the auricles, hence the greater the block

As regards the ventricle, the greater its irritability and rhythmicity the sooner it will respond to its own internal stimuli with a contraction and the shorter the stoppage and the more rapid the rhythm. A low ventricular rhythm (under 25 per minute) is therefore often a sign of poor condition of the ventricle and of a tendency to stoppage during the period of complete block in spontaneous attacks.

The experiments of Erlanger and Hirschfelder have been confirmed by v. Tabora under Hering's direction. The latter has found that stimulation of the vagus may under certain circumstances increase the degree of heart-block and facilitate the onset of stoppage, especially when digitalis has been administered. The apparent discrepancy between their findings is probably due to the presence of the different nerve-fibres in the vagus, so that sometimes conductivity, sometimes irritability is most affected

RELATION OF HEART-BLOCK TO ADAMS-STOKES SYNDROME.

It cannot be too strongly emphasized that (1) heart-block (complete) and Adams-Stokes syndrome are by no means synonymous; the former represents merely the dissociation of rhythms, while the Adams-Stokes syndrome brought on by cerebral anæmia during ventricular stoppage is a totally different matter; (2) heart-block may persist for months or years without the occurrence of the syndrome, as in the case about to be described.

Attacks of the Adams-Stokes syndrome may occur in three ways. (1) at the transition from normal rhythm to complete block; (2) in the midst of complete block; (3) probably also from stimulation of the vagus in certain cases where conductivity is already diminished.

In the cases where the Adams-Stokes syndrome (ventricular stoppage) appears at the transition from normal rhythm to complete block, the attacks are usually preceded by quickening of the pulse; and the block passes off and reappears suddenly. When the complete block becomes permanently established, the Adams-Stokes syndrome may disappear, as is well shown by the following case,[1] seen by the writer in consultation with Dr. H. G Beck. The Adams-Stokes syndrome may be present only in the initial and not the later stages of the heart-block.

CASE OF HEART-BLOCK, WITH ADAMS-STOKES SYNDROME ONLY AT ONSET OF BLOCK.

J. L., aged 72, had been perfectly healthy all his life except for attacks of malaria when between 14 and 40 years of age, and pneumonia about ten years ago. Denies syphilis and gonorrhœa; drinks little, but smokes considerably. Has been a blacksmith until July, 1907. At this time he was struck on the head by a railroad gate, became unconscious for one or two minutes, after which he recovered at once except for a slight transitory weakness of the right arm and slight transitory aphasia. He remained well until November, 1907, pulse being 60 to 64. In the latter part of November he began to have weak spells in which he fell but did not lose consciousness. He was seen by the writer on January 12, when he had been having numerous attacks for about a week.

FIG. 273.—Tracing from jugular vein and carotid artery in a case (J. L.) of complete heart-block after the syncopal attacks had subsided.

Patient was a fairly nourished man of good rosy color, pupils reacting normally; no signs of intracranial disturbance nor of lues. Chest clear. Heart not enlarged, action regular, pulse rate 33 per minute. Sounds accompanying the beats are loud and the first sound is accompanied by a flowing systolic murmur not transmitted to axilla. Second sound clear. Between these in the long pause there can be heard two or three very soft distant sounds like the ticking of a watch accompanying which small undulations may be seen over the jugular vein and on most delicate palpation of the radial a slight impulse can be felt there as

[1] A study of this case has been reported by Drs. H. G. Beck and W. R. Stokes, Arch. Int. Med., Chicago, 1908.

well, due to the beating of the auricles against the root of the aorta. The venous tracing (Fig. 273) showed complete heart-block. At this time he had no attacks. On January 17, however, he was again seen. His attacks had been very numerous, the pulse-rate rising and falling with great rapidity. Tracings from the jugular vein and carotid arteries, taken as an attack came on, show the following sequence of events. At first a period of complete heart-block lasting a few minutes. This then passed off and was succeeded by a few moments of 2 : 1 rhythm. The 2 : 1 rhythm passed suddenly into a 1 : 1 rhythm at a rate of about 90 per minute and began to quicken. It was then prophesied that an attack was imminent, and in an instant the ventricles suddenly ceased to beat. The patient cried out, became ashy pale, and a convulsion set in, during which the auricles continued to beat at the old rate. In about 11 seconds the ventricles began to beat, and soon resumed their regular independent

FIG. 274.—Diagram representing the conditions found in the tracing Fig. 273. *A*, auricular contractions; *V*, ventricular contractions; 1, 2, first and second heart sounds; *a*, *a* sounds due to contractions of the auricles.

rate of 28 to 30 per minute in complete block. After a few more seizures an hour or so passed without further change in rate or further symptoms. When seen in the afternoon and again on the next day the pulse-rate had not varied. It was then prophesied by the writer that no more attacks were imminent, but the patient was kept in bed for several weeks afterwards. A few weeks later he had a sinking spell with weakness of the pulse, but no change in rate and regularity and no unconsciousness or convulsions. Thus he also recovered from and remained free from symptoms and attacks until his death two months later. He died rather suddenly but was conscious to the last; his pulse-rate had not changed, and he died not from the Adams-Stokes syndrome but from his coronary sclerosis. The lesion found in the His bundle will be discussed below. In this patient the Adams-Stokes syndrome passed off as the complete block became established.

Variations in Pulse-rate. This case is no isolated example of such a condition. Even Burnett's case (1824) furnishes an example, for he says, "the pulse beats at the rate of 71 in the minute for the space of about a minute, then intermits for 7, 8, or 10 seconds . . . In the evening I found that he had been attacked many times but was then much better . . . He complained, however, of more pain about the precordia and his pulse beat only 20 in the minute." A similar tendency to improvement after heart-block set in has been noted in Stokes's first case (1846), in that of Alfred Webster (1900), in one of the cases reported by Edes (1901), and in one of Erlanger's cases (1905).

Stoppage of the Ventricles during Complete Heart-block. Unfortunately, the Adams-Stokes syndrome does not always end with the establishment of permanent complete block. Just as in the two experiments of Erlanger and Hirschfelder mentioned above, stoppage of the ventricles sometimes occurs in the midst of a complete block when the pulse-rate is slow and without preliminary variations in rate. This took place in the case reported by His and in Erlanger's first case. The influences producing this stoppage act directly upon the ventricles, the auricular rate being

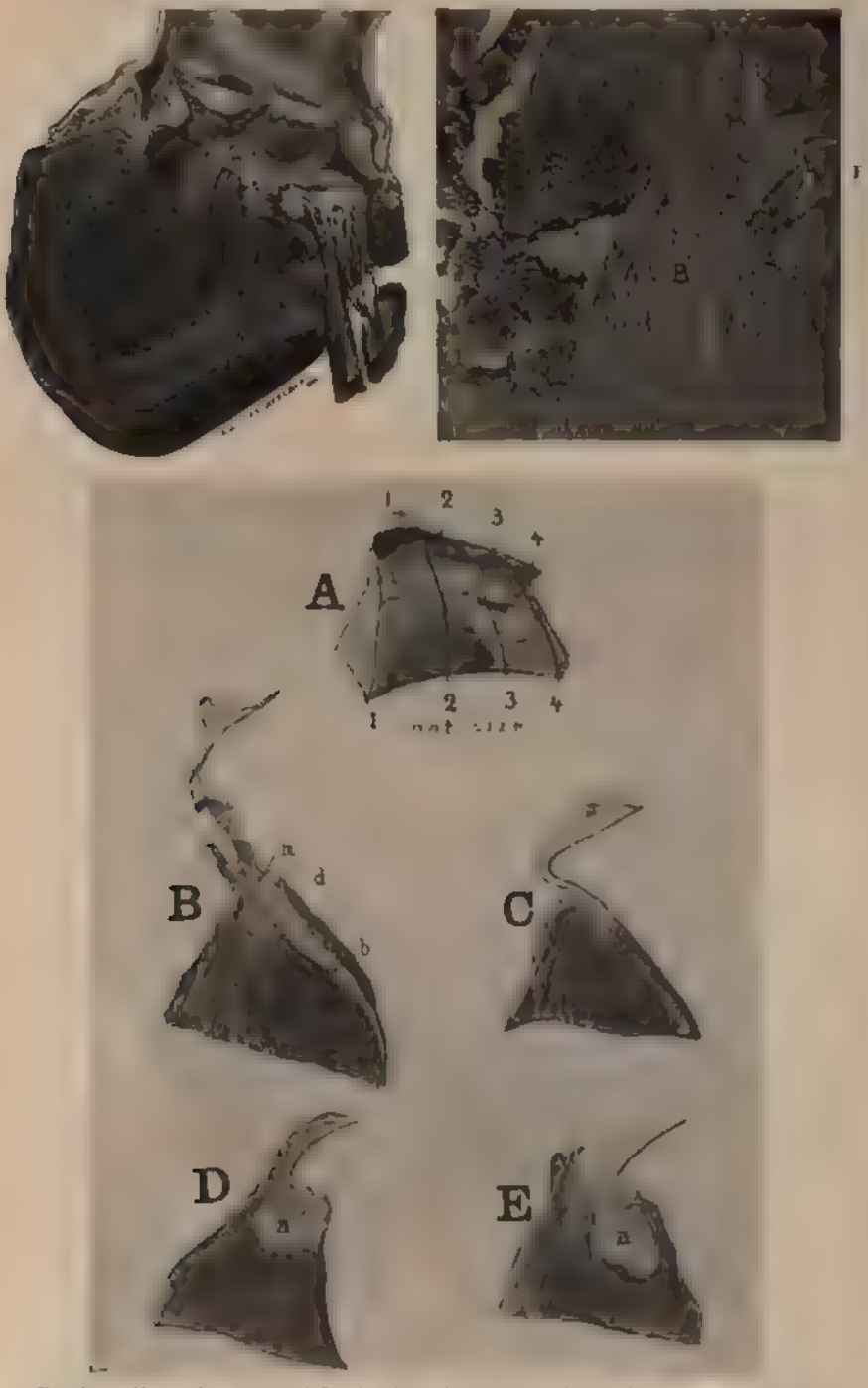

FIG. 275.—Heart of a patient (J. L.) showing calcifications which produced Adams-Stokes disease. (Drawn from the specimen.) *A, B, C, D, E*. Sections through the interventricular septum, showing the calcification pressing upon the auriculoventricular bundle. After Beck and Stokes. *F*, section through a. (Photomicrograph by Dr. C. S. Bond.)

unchanged or quickened, but the nature of these influences is not well understood. Erlanger has shown that the plugging of a coronary artery in animals does not bring stoppage from complete block. On the other hand, slight asphyxia, such as holding the breath after slight exercise,

FIG. 276.—Diagram showing the two types of ventricular stoppage producing the Adams-Stokes syndrome. 1. Ventricular stoppage only at the moment when conduction ceases. 2. Stoppage of the ventricles setting in during the periods of complete block. A, auricular contractions, V, ventricular contractions, A–V, conduction of impulses from auricles to ventricles.

brought them on regularly. However, it cannot be said that the prognosis is much if any more unfavorable in these than in the other group of cases, since Erlanger's case at least lived several years after observation, and this point has not been noted in most of the reported cases. Prof. Thayer has recently reported a case in which the block has passed off.

LESIONS OF THE AURICULOVENTRICULAR BUNDLE.

These two groups represent cases in which the block appears to be myogenic; and pathological evidence indicates that such is the case. Although Adams (1827) mentions fatty degeneration of the ventricular septum in his case, and many other autopsies had been performed, the first case in which a lesion of the auriculoventricular (or atrioventricular) muscle bundle was demonstrated was that of Luce in 1902 in which a sarcoma was found involving the auriculoventricular bundle. Luce, however, did not regard this as a causal factor for the Adams-Stokes syndrome, and the first case in which this connection was definitely established was reported by Stengel, of Philadelphia, in 1905 with excellent figures showing fibrosis of the bundle. Soon after this Schmoll reported a case in which no lesion could be discovered macroscopically, but fibrosis of the His bundle was demonstrated with the microscope.

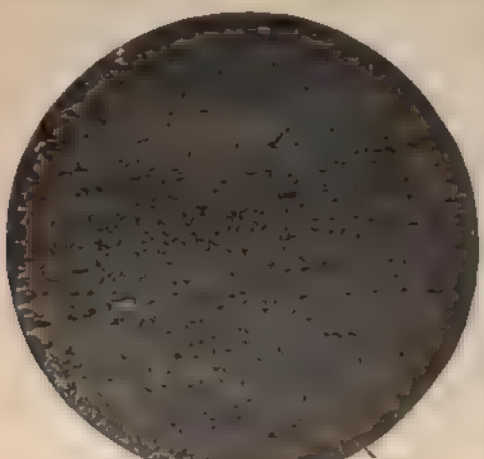

FIG. 277.—Section of a luetic infiltration of the auriculoventricular bundle. After Aschoff, Norris and Cavenson. INF, area of round-cell infiltration.

In the case of J. L. reported above, autopsy showed extensive atheroma of the aorta; the coronary arteries were converted into pipes of bony hard-

ness. Large calcifications were present upon the mitral valve and in the upper part of the interventricular septum, in which a long tongue of calcification can be seen to intercept the auriculoventricular bundle. This was beautifully shown in the sections which were made by Stokes under Retzer's direction, and thoroughly explain the clinical features observed.

In the past two years a considerable number of cases have been studied both histologically and physiologically, lesions in the His bundle being uniformly found. The following represent some of the lesions reported

Gumma, 7 Handford (1904), Keith and Miller (1906), Grunbaum (1906), Ashton, Norris, and Lavenson (1907), Heneke (1907), Fahr (1907), Rendu (1895)

Calcified patches involving the bundle, 4 Stengel (1905), Hay and Moore (1906), Beck and Stokes (1908), Heneke, Muller, and Hoesslin (1908)

Fibrosis of the bundle, 6 Schmoll (1906), Gibson, G. A. (1906), Fahr (1907), Gibson, A. G. (1908), Dock, G., Vaquez and Eamein.

Tumors in the septum Fibroma Sendler (1892) Round-celled sarcoma Luce

Anæmic infarction of the auriclo(atrio)ventricular bundle. Jellinek, Cooper, Ophuls (1906), MacCallum (1908).

Simple round-celled infiltration of the auriculoventricular bundle, 1. Heneke, Muller, and Hoesslin (1908).

Mural ulceration involving the auriculoventricular bundle (ulcerative endocarditis) James (1908).

Fatty degeneration: Butler (1907).

Arteriosclerosis of artery supplying auriculoventricular bundle: D Gerhardt (1908).

Absence of demonstrable lesion, macroscopic or microscopic. Heneke, Muller, and Hoesslin (1908).

CASES OF ADAMS-STOKES SYNDROME NOT DUE TO LESION OF THE AURICULOVENTRICULAR BUNDLE.

Although the overwhelming majority of cases of the Adams-Stokes syndrome (persistent bradycardia, complete heart-block, and syncopal attacks) have been proved to be due to lesions of the auriculoventricular bundle, a few cases in the literature remain which must still be regarded as due to over-stimulation of the vagus.

The most typical of these attacks is described by Thanhoffer (1875). A colleague was compressing his own vagi in the neck, when suddenly he "stared at me with glassy eyes, without releasing his grip and without answering. I could remove his hand from his throat only with the greatest force and they still remained clenched. Consciousness did not immediately return even after removing his hands."

Another case was reported by Neuberger and Edinger in 1898. The patient was a neurasthenic man, aged 46, who had been repeatedly examined by various physicians but no signs of organic nervous disease discovered. He suffered from severe constipation. From Nov., 1896, to January, 1897, he occasionally fainted when at stool. His pulse during that period was usually 60 between attacks. On January 1, 1897, he fell in a faint while having a desire to go to stool, his head and eyes were drawn to the left and the eyes twitched. During that day he had several similar attacks, before each of which the pulse disappeared, returning during the attack to a rate of 16 to 18 per minute. By evening the rate had returned to 60. He died in one of these attacks on January 8. Autopsy performed by Carl Weigert, showed almost complete atrophy of the right half of the cerebellum and a varicose dilatation of the ependymal vessels in the medulla. It is probable that at stool or during effort the pressure in these varices rose and caused them to compress the medulla near the vagus nucleus. In spite of a very careful search by Weigert, no myocardial lesion could be found. It is, therefore, fair to assume that in this case the attacks and the probably existing heart-block were vagal in origin.

Dr. Walter James also reported a case in which recurrent groups of ineffectual extrasystoles caused the circulation to become so slow at times as to produce syncopal attacks.

A somewhat similar case was that of Holberton (l.c.) (1841), in which the attacks dated from a fall from a horse, and no myocardial lesion was found. On the other hand, our case (page 467) illustrates the need of caution in reaching this conclusion, since there the attacks dated from a blow upon the head, and yet autopsy proved the presence of a most typical lesion of the auriculoventricular bundle. Since these lesions may be microscopic (Schmoll, Gerhardt), it is evident that a neurogenic origin of the syndrome can be diagnosed only when the bundle has been examined by serial sections. The writer has also seen in consultation a case of complete heart-block associated with a tumor along the course of the vagus.

Since it requires a very considerable lesion to produce the neurogenic syndrome, and since death occurs from the latter cause as well as from the syndrome, the prognosis is no better in these cases than in the myogenic, except in cases where the causal factor (tumor, etc.) may be removed by operation or by treatment. However, it must be added that even with the most liberal interpretation neurogenic cases are extremely rare, and the presence of the Adams-Stokes syndrome may almost always be regarded as *prima facie* evidence of a lesion of the auriculoventricular bundle.

Atropine Test.—The origin of the block in these cases can be readily demonstrated by paralyzing the vagi by the administration of atropine, 1 mg. ($\frac{1}{65}$ gr.), which causes the block to pass off in the neurogenic cases and the pulse-rate to become rapid, but does not affect it in the myogenic. In most cases of the typical Adams-Stokes disease (Edes, Erlanger, Schmoll), atropine does not affect the rate, and the organic nature is further proved. In our own two rather exceptional cases the atropine test was, unfortunately, not permitted. Gibson and Ritchie have reported a most interesting case in which both neurogenic and myogenic factors seemed to be at work, since the complete block disappeared promptly upon giving atropine and reappeared an hour later when the effect had worn off. However, conductivity was always diminished (conduction time, *a c* interval, being 0.6 second instead of 0.2) even when the vagi were paralyzed, so that the auriculo-(atrio)ventricular bundle was probably injured as well. Professor Thayer and Dr. F. W. Peabody have found that atropine caused the partial block (4 : 1 rhythm) to pass off, but had no effect when the block was complete. This observation harmonizes well with the other experimental and clinical evidence regarding complete and partial blocks.

CONTRIBUTING FACTORS.

The Adams-Stokes syndrome is more common in men (84 per cent. of Edes's cases), of which 48 per cent. occurred between the ages of 50 and 70. This, as well as the autopsy series mentioned above, establishes the importance of coronary sclerosis as an etiological factor. It is quite striking that in two cases of this small series (that of Cooper, Jellinek, and Ophuls and one of those mentioned by Dietrich Gerhardt) gonorrhœal infection was the etiological factor. Mackenzie describes cases of partial heart-block (2 : 1 rhythm) as a result of influenza and pneumonia, and the writer has seen a similar depression of conductivity during the course of the latter. Powers has reported a case with partial halving of the rate after pneumonia. Saigo has found extensive vacuolization, fatty and parenchymatous degeneration, and cellular infiltration in the Purkinje fibres of the conduction system following acute rheumatism and other diseases.

These were especially marked in the left branch of the auriculoventricular bundle. The importance of infectious diseases as directly causal factors is further shown by the case of Butler, in which the bradycardia dated from an attack of typhoid fever, and also in Dunn's case, in which a radial pulse (18 per minute) and typical Adams-Stokes syndrome occurred in a boy of 11 on the ninth day of diphtheria. There can be no doubt that many of the sudden deaths from pneumonia and diphtheria are due to heart-block.

Cardiac overstrain may also be a factor, as in the case of a boy of 15 reported by Strubing, who when otherwise healthy ran a considerable distance, fainted, then walked home, and had several other syncopal attacks with convulsions. His heart was enlarged, and the pulse 16 to 18 per minute. Rest and proper treatment brought about some improvement, and his pulse finally rose to 44, but he died soon afterwards.

It is possible that ptomaine poisoning or autointoxication due to severe gastro-intestinal upset may give rise to the syndrome.

Case of Adams-Stokes Disease with Subsidence and Recurrence of Symptoms, and with Attacks during Complete Block.

Recently the writer, with Professor Thayer and Dr. H. M. Thomas, examined a gentleman who had lately suffered from a severe acute gastro-enteritis with vomiting and severe diarrhœa lasting several days. During this time he had fainted several times while at stool and his physician found him with a pulse-rate of 20 per minute. With the improvement in digestion this bradycardia passed off within a few days, and he had no further syncopal attacks. When examined ten days later his pulse-rate was 60, increasing normally upon slight exercise. There was no sign of heart-block, and conduction time (a–c interval) was normal (.02 second). No signs of nervous disturbance were present. After a few weeks of good health the fainting spells and bradycardia returned, and lasted for several months. Prof. Thayer informs the writer that during this period the rhythm varied from 1:1 to 2:1 and 4:1, returning to normal rate when atropine was administered. Syncopal attacks also occurred frequently in the midst of complete heart-block, and during the periods of complete heart-block the rate was very slow and irregular. In complete block the rate was unaffected by atropine.

After some months conductivity gradually returned, and at the time of writing the patient has remained quite well and has had a normal pulse-rate for several months.

The presence of a hemorrhage in the auriculoventricular bundle or its vicinity (apoplexy of the bundle) would account for the occurrence and the subsidence of these symptoms. An infiltration or fatty degeneration of the bundle might account for the occurrence and subsidence of the first attack, but scarcely for the sudden recurrence during a period in which the patient had been in excellent health.

Prentiss also mentions a case brought on by heavy lifting, in which either hemorrhage or myocardial degeneration may have been the cause.

Partial heart-block has been reported from overdoses of digitalis (Mackenzie, Hewlett, A. G. Gibson) (page 179), but these have never given rise to complete block or Adams-Stokes syndrome.

PHYSICAL SIGNS AND DIAGNOSIS.

As the Adams-Stokes syndrome may occur in cases having valvular lesions, the physical signs over the heart may vary, and all forms of murmurs and of cardiac insufficiency may occur. Those which are character-

istic of the condition, as observed by Stokes and Chauveau, are the very slow pulse disappearing entirely before the onset of the attack; the presence of small visible pulsations in the jugulars, of more than double the number of the pulse in the carotids, with the small jugular pulsations and occurring at a regular rhythm which is more rapid than that of the ventricles; a faint sound like the ticking of a watch may often be heard near the left sternal margin, i.e., the right auricle; and a slight pulsation or shock may at the same time be seen or felt over the apex. On most careful palpation a faint shock of the same rhythm may also be felt in the radial pulse. This corresponds to the small auricular wavelets upon the pulse, which, as François-Franck has shown, are due to the beating of the auricles against the root of the aorta.

All these signs may usually be made out in cases of heart-block, and the diagnosis should therefore be made by any clinician in the ordinary physical examination.

In X-ray examination the independent contraction of the auricles may be readily seen (Deneke), and this of course settles the diagnosis. Similarly G. A. Gibson and Einthoven, as well as Barker, Bond, and the writer, have demonstrated heart block by the electrocardiogram.

The usual and the most satisfactory method of diagnosis is by comparison of the venous pulse tracing with that from the carotid artery or the apex, by which means the exact relation of auricular to ventricular contraction, the degree of block, and the variations of conductivity are readily shown.

Difficulties in diagnosis may occur from the following causes:

1. The pulse-rate may be so slow that heart-block may be suspected. This occurs especially in old persons, in athletes when at rest, and in convalescents from infectious diseases. For the absolute exclusion of heart-block a venous tracing may be necessary, in which the absence of a wave midway between the normal *a* waves excludes the presence of a heart-block. The writer has seen a number of cases whose pulse-rate was 44 to 48 per minute with no sign of heart-block on the venous tracing. (Figs. 48 and 106.)

2. The early diastolic wave (*h* wave of Hirschfelder, *b* wave of Gibson) may sometimes occur midway between auricular waves, and may thus simulate a 2 : 1 rhythm. Moreover, the "third heart sound" is usually present in these cases, and may easily be taken for the sound of auricular contraction. The presence of the *h* wave may be differentiated from that of partial block by increasing the heart-rate, by rapid respiration, mild exercise, etc., upon which the *h* wave is no longer found midway between *a* waves, but maintains its old interval from the *c* wave and approaches the second *a*, whereas in partial heart-block it would maintain the mid-position. This point is of great importance, since the presence of heart-block is a grave sign, and it should not be diagnosed without due care. The writer has seen a number of cases in which heart-block might have been diagnosed had this precaution not been exercised, as for example the patient whose phlebogram is shown in Fig. 106.

Slow pulse of vagal origin may also occur in brain tumor, fracture of the skull, meningitis, etc., and, especially in the first, may be

accompanied by syncopal attacks. In these cases there is rarely any degree of heart-block between attacks, and the site of the cardiac disturbance can readily be determined by its disappearance after the administration of atropine.

A slow pulse with occasional attacks of vertigo may also occur as the result of extrasystoles too weak to open the aortic valves, and thus give rise to a rhythm which is too slow to nourish the brain (W. James), and a true Adams-Stokes syndrome arises without heart-block. The diagnosis is, however, readily made on auscultation, from the fact that between effective beats a single loud sound is heard (whole rhythm being lub dub lub, pause, ... lub dub lub) and not the feeble ticking auricular sound of auricular contractions. The venous pulse and electrocardiogram characteristic of extrasystoles (see page 68) establish the diagnosis.

In occasional cases, paroxysms of tachycardia are accompanied by fainting spells, the pulse between attacks being quite normal or even very slow.

Case of Paroxysmal Tachycardia with Syncopal Attacks, Suggesting Adams-Stokes Disease.

A few years ago the writer examined such a case in consultation with Professor Barker and Dr. I. P. Lyon of Buffalo. The patient was a man past middle age, had a pulse-rate of 60, and had been subject to attacks of palpitation with fainting spells. The case had been seen by several specialists in various cities, who had diagnosed it Adams-Stokes syndrome. Physical examination was negative except for a slight grade of arteriosclerosis. Tracings of the venous pulse, however, showed conductivity to be normal (a-c interval 0.2 second), and this continued to be the case even when, upon exercise, the pulse-rate rose to 120 per minute without dropping a beat. The Adams-Stokes syndrome was thus excluded. From the sudden onset and the fainting spells during the attacks, it was concluded that the condition was most probably paroxysmal tachycardia. The patient was subsequently seen in a typical attack, with sudden approximate doubling and sudden halving of the rate, and the diagnosis was thus verified. Dr. Lyon informs the writer that the patient is much improved and has now only mild attacks of tachycardia.

There may occasionally be difficulty in differentiating the Adams-Stokes syndrome from epilepsy and brain tumor. Heart-block is, however, never present in the former, very rarely in the latter, so that the diagnosis can usually be made from simple inspection of the jugular vein. If necessary, venous tracings, supplemented by the atropine test, may be resorted to.

Prognosis.

The course of cases suffering from the Adams-Stokes syndrome is very variable. It is probable that many cases die in the first attack, but the condition remains undiagnosed or is ascribed to coronary sclerosis. It is not unlikely that histological examination of many cases of sudden death would reveal lesions in the bundle of His or its artery. In some cases death occurs within a few weeks or months after the first attack, but in very many the heart-block may last for many years, with or without disappearance of the syncopal attacks. Many cases of Edes's series lived seven or eight years after the first attack. Osler's case lived thirty years after the onset of bradycardia, and seven years after the first syncopal attack.

It is stated that the pulse-rates of Julius Cæsar and of Napoleon were abnormally slow (Napoleon's being sometimes 40, but at Elba 50 to 55), and that the epilepsy of the latter was a sign of the Adams-Stokes syndrome, but this is not proved. However, it is certain that the presence of complete heart-block is compatible with ability to do a considerable amount of work. Vigouroux had under observation a laborer with complete heart-block who during five years did hard work, driving a cart with six oxen in the hottest weather. His heart always beat at a rate of 20. Dr Archibald Hewan was able to climb a mountain several thousand feet high when his pulse ranged from 32 to 40 and never rose above the latter. Most of the cases die in the attacks (Edes), but death from coronary sclerosis without Adams-Stokes syndrome, as in our case, is not uncommon. Gerhardt has recently reported three cases in which not only the syncopal attacks but also the heart-block completely disappeared, owing to subsidence of the pathological process in the auriculoventricular bundle which was not totally destroyed. Prof. Thayer's case, quoted above, probably belongs to this group.

TREATMENT.

As regards treatment it must be frankly admitted that there is no drug at our disposal which either improves conductivity, prevents stoppage of the ventricles, or increases the ventricular rate.

General experience has shown that *digitalis* is either without effect or *positively harmful*, and v. Tabora has shown experimentally that it both decreases conductivity and increases the period of stoppage.

Caffeine, theobromine, strychnine, strophanthus, and amyl nitrite are equally without effect upon either of these phenomena in both clinical usage and, as the writer has found, in experimental heart-block in dogs. *Atropine* is usually without effect, but may be of temporary benefit in certain cases with a neurogenic element (as in those of Gibson and Ritchie and Thayer). August Hofmann reports a case in which inhalations of *oxygen* were of distinct benefit, but in the writer's experience this is not often the case. Ammonium carbonate, with which Burnett claimed to have aborted attacks, has not been much used in recent years.

In the *syphilitic cases*, however, antiluetic treatment — *inunctions of mercury* and administration of large doses of *potassium iodide*, ascending to 4 Gm. (℥j) t.i.d., by mouth—has been known to bring about a cure by absorption of the gummatous lesion (Schmaltz, Erlanger), and should be tried whenever there is a suspicion of lues.

Peculiar *postures* sometimes help in warding off syncopal attacks by improving cerebral blood flow until the ventricular rhythm has become established. Stokes (1846) writes that his patient "had two threatenings of fits since his admission, and warded both off by a peculiar manœuvre: as soon as he perceives symptoms of the approaching attack he directly turns on his hands and knees, keeping his head low, and by this means he says he often averts what otherwise would end in an attack." This was the patient's physiological therapy to prevent cerebral anæmia.

It is evident that we have no specific remedy or procedure for the relief of the Adams-Stokes syndrome. Nevertheless, since most cases live several years after the first attack, a good deal can be done for the patient. The two aims to be kept in view are, first, to avoid anything which brings on rapid changes of pulse-rate (excitement, emotion, exertion, straining at stool), and, second, to enable the ventricles to gain their inherent rhythmicity and thus obviate stoppage. As has been seen from Schuster and Prentiss's case and from the effect of exercise in Erlanger's case, exercise is a particularly potent factor in bringing on attacks in the early stage of the disease. Absolute rest is therefore indicated as long as the pulse-rate is at all unstable (i.e., when there is transition from partial to complete block) and *vice versa*.

When complete block has become permanent (for at least several weeks) the condition is quite different. The ordinary influences (emotion, mild exercise, etc.) affecting auricular rate now no longer play a rôle, and the attack seems to be chiefly due to the action of CO_2 upon the ventricular muscle, as in holding the breath after exercise, or in the similar phenomena when straining in order to lift, void, or defecate. In the stage of complete block the patient may therefore be gradually allowed exercise which does not cause him exertion. He must learn to avoid the latter, since it may bring him sudden death.

In all stages of the disease the bowels should be kept open and the stools fluid, but excessive purgation and straining at stool should be avoided.

Moreover, it should not be forgotten that in a certain number of cases, like that of J. L. (page 167) and those of Gerhardt mentioned above, the entire process may be due to acute toxic or myocarditic changes and may be transitory, so that if the patient be kept at rest during the acute period the whole inflammatory process in the auriculoventricular bundle may subside and perfect health may return, whereas strain upon the heart may prevent the inflammatory process from subsiding and may cause the attacks to continue.

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XII.

PERICARDITIS.

Historical.—The presence of changes in the pericardium in animals was known to Galen, and Senac in 1749 described the condition in man. Auenbrugger and later Corvisart were able to make out changes in dulness due to pericardial effusions. Laennec detected the murmur of fibrinous pleurisy, and described it as resembling the creaking of a new saddle, but its diagnostic significance was positively established by Collin and by Devilliers in 1824.

ETIOLOGY.

The frequency with which pericarditis occurs varies greatly according to various observers, and particularly according to the age of their patients. It seems to be considerably more common in children than in adults with cardiac disease, as stated by Poynton, as it accompanied endocarditis and myocarditis in 94 per cent. of Sturges's cases of heart disease (carditis) from the Great Ormond Street Children's Hospital. This is in accordance with the somewhat exaggerated statement of Cadet de Gassicourt that all children who are killed by rheumatism die from pericarditis; but it applies more to children of the second decade than of the first, since death from rheumatic affections is most common in the second decade. Pericarditis was found in 19 of Osler's 73 autopsies upon cases of chorea. Pericarditis occurred in 230 (1 per cent.) of the cases admitted to the medical service of the Johns Hopkins Hospital. Of these 53 were associated with endocarditis; 8 with myocarditis. Other factors were pneumonia 39; rheumatism 31; nephritis 33, tuberculosis 25, pleurisy 17, gonorrhœa 3; aneurism 2, leukæmia 2, syphilis 1. Rheumatism occurred in 51 per cent. of the 100 cases reported by Sears from the Boston City Hospital. This relationship between pericarditis and rheumatism has been proved experimentally by Wasserman, Triboulet, Poynton and Paine, Walker, Cole and Beattie (page 301).

Pneumonia is also one of the common causes of pericarditis (18 per cent. of Sears's series), and usually ranks next to the rheumatic cycle as an etiological factor. Pericarditis was present in 4.66 per cent. of the cases of pneumonia at the Johns Hopkins Hospital (Chatard), and in 2-3 per cent. of Preble's series in Chicago. According to the latter observer its relative frequency is about proportional to the extent and severity of the disease. This claim is also borne out by Chatard's statistics (frequency of 15.7 per cent. in the cases coming to autopsy). Moreover, the appearance of an acute pericarditis in the course of the disease is a very grave sign, for only two cases (6.5 per cent.) of Chatard's series recovered.

Pericarditis is also common in scarlatina (especially with streptococcus infection), in severe measles, and in smallpox. In the latter it is frequently purulent.

Tuberculous pericarditis is quite common (8 per cent. of Breitung's autopsies), and in contrast to the rheumatic form rarely subsides. It often ends in effusion.

The pericarditis of chronic nephritis and uræmia constitutes a frequent termination of this disease, though it is by no means always fatal. It is usually due to an intercurrent infection, and the pyogenic cocci can often be cultivated from the exudate.

Pericarditis may also result as a secondary infection in septicæmia and in puerperal infections as well as in gonorrhœa, especially when there is accompanying arthritis. It is rare in typhoid fever (3 times in McCrae's 1500 cases); occurs occasionally in influenza, and sometimes results from septicæmias due to B. coli, B. aerogenes capsulatus, B. pyocyaneus, etc.

Trauma without direct injury of the pericardium or viscera was the cause of pericarditis in Blaneard's case in 1688, and a large number of cases due to this cause were collected by Bernstein in 1896. Blows upon the chest, wagon running over the body, etc., are the common causes.

FORMS OF PERICARDIAL EXUDATION.

The exudate into the pericardial cavity may assume various forms. In simple venous stasis and asphyxia of the endothelial cells (*hydropericardium*) a clear thin fluid of low specific gravity, relatively poor in proteid and especially in fibrinogen, is secreted. When there is true inflammation of the pericardium, the exuded fluid is rich in fibrinogen and of relatively high specific gravity (over 1015) and contains nucleo-albumen (clouding with acetic acid). Samuel has shown that when the exudate is poor in fibrin ferment it remains fluid (pericarditis with effusion), whereas when this is present the fibrinogen coagulates (*fibrinous pericarditis*). According to Opie, the enzymes are derived chiefly from the leucocytes, especially the polymorphonuclears, and hence the amount of fibrin deposited depends largely upon the number of these cells present. Moreover, since these cells pass out from the blood-vessels, the fibrin is first and most thickly deposited in the vicinity of the latter.—*i.e.*, along the epicardium above the circumflex and descending rami of the large coronary arteries, where it begins in the form of strands passing out from about the leucocytes, and hence gives the heart a shaggy appearance (cor villosum, Fig. 279). This layer of fibrin usually has the appearance and consistency of a yellow butter. When fresh it is not very adherent to the heart, and may reach a thickness of an inch or more. There may be no fluid in the pericardial cavity, but, as a rule, both fibrin and fluid are present, the latter often in large quantities. The fluid is usually thick, containing uncoagulated fibrinogen as well as small flaky masses of fibrin, which may render it too thick to be removed by aspiration. When the exudate is extremely rich in bacteria and leucocytes, the proteolytic enzymes are given off, which digest the fibrin, and the fluid becomes purulent.

When the fibrinous exudate of a simple pericarditis is absorbed rapidly, it leaves no traces and the pericardium again becomes clear. But when it

lasts for some time and the resolution is slow, organization takes place, and white patches of pericardial thickening ("milky spots") are found over the surface of the heart.

These may, however, result from small perivascular foci like those of chronic myocarditis, without ever giving rise to the clinical picture of pericarditis.

FIG. 278.—Acute fibrinous pericarditis.

FIG. 279.—Tuberculous pericarditis (cut villous).

Organization and Adhesion.—The strands of newly formed connective tissue may penetrate the fibrin between the two layers of pericardium and completely bridge the cavity with fibrous strands (Fig. 286). In many cases the tug of the heart in systole stretches these out into fibrous cords an inch or more in length; in other cases, or over other parts of the same heart, the adhesions are denser, the two surfaces may be completely glued together and the cavity obliterated (adherent pericardium).

The division into these forms of pericarditis is, therefore, an arbitrary one, but, as will be seen, is made necessary by the absolute difference in both diagnostic signs and mechanical effects upon the circulation, and thus as regards indications for treatment. Their relative frequency is shown in the following table, which Gibson quotes from Breitung's autopsies at the Berlin Charité Hospital (Virchow's Department).

	Cases.	Per cent.
Serofibrinous	108	33 3
Hemorrhagic	30	9 2
Purulent	24	7 5
Tuberculous (secondary)	24	7 5
Tuberculous (primary)	2	0 7
Partially adherent	111	34 3
Totally adherent	23	7 3
Ossified	2	0 7
	324	100

Although the pathogenesis is the same, the clinical manifestations of fibrinous pericarditis, pericardial effusion, and adherent pericardium are different; hence they are discussed separately.

SIMPLE FIBRINOUS PERICARDITIS.

PATHOLOGICAL PHYSIOLOGY.

The friction due to the presence of the fibrinous exudate imposes a slight increase in the resistance to both contraction and filling of the heart. The exudate itself takes up a certain amount of space in the pericardial cavity and may thus somewhat diminish the filling of the heart, but these factors rarely suffice to embarrass the circulation.

Either as a result of the accompanying injury to the heart muscle or from irritation of the depressor nerve, the peripheral vessels are dilated and the blood-pressure is low. The pulse also becomes small and rapid, but is usually regular.

SYMPTOMS.

Precordial pain, palpitation, shortness of breath, and weakness are the common complaints, as well as occasional chilly feelings. Fever, with which these are associated, is generally, but not always, present.

The onset is very often insidious, and the disease may not be recognized at all by the patient. *Precordial pain* is the most striking symptom. Sibson estimates that it occurs in 70 per cent. of the cases. Henry Head calls attention to the fact that the pain of pericarditis is not a referred pain, but a true local pain, often limited to the area over which the friction is audible and associated with tenderness on pressure and on percussion. It does not radiate from this site, and differs in this respect from the anginal pain. The other symptoms, shortness of breath and palpitation, manifest no special peculiarity

Occasionally, especially when the pericarditis affects the posterior wall of the pericardium, there is *pain on swallowing*. This pain is in every way similar to the tenderness of the interspaces in front, and occurs when the bolus of food presses upon the pericardium as it passes down the œsophagus.

When the recurrent laryngeal nerve is affected by the inflammation, *aphonia* or change in the voice results. Involvement of the phrenic often produces *hiccough*.

PHYSICAL SIGNS.

The patients are usually quite pale, occasionally cyanotic. Except for accompanying joint involvement, fibrinous pleurisy, or pulmonary consolidation, there are few signs outside the heart. Œdema of the extremities is rare unless there are accompanying valvular lesions. Over the heart there may be some precordial bulging, especially in children, but the cardiac impulse may be less marked than usual, weak, diffuse, and wavy. On palpation there is sometimes a slight superficial scratching felt, especially between the left parasternal line and the sternum, but this is by no means as marked, as frequent, or as regular as in valvular lesions.

The area of cardiac dulness and flatness may or may not be increased in one or both directions, dependent upon the amount of the exudate as well as upon the degree of dilatation of the heart, but the outlines characteristic of pericardial effusion are not present when the exudate is plastic.

The pathognomonic sign of fibrinous pericarditis is the *superficial scratching or churning murmur or friction sound* described by Laennec as resembling the rubbing of a new saddle. It can be imitated more or less closely by placing the palm of the hand over the ear and then scratching to and fro upon the back of the hand with the finger-nail. The pericardial friction is exactly similar in character to the friction heard in pleurisy, but its time is coincident with the cardiac cycle. It does not, however, coincide sharply with either systole or diastole, but is usually heard during portions of both. It is usually louder during systole than during diastole, probably because the two surfaces are moved across one another with greater force. A short pause usually occurs between the systolic and the diastolic portion of the friction. The diastolic friction is softer than the systolic, occurs rather early, and ceases during the latter half of this period, or in other words as ventricular filling diminishes. It may also be heard again at the time of auricular systole, giving a triple sound to the friction (Broadbent). Sometimes, especially when the pericarditis is just beginning and the friction very soft, it is not heard at all during diastole.

Fig. 280.—Diagram showing the relations of the pericardial and pleural frictions to the cardiac and respiratory movements. The pericardial friction is indicated by narrow zigzag line, the pleural friction by black triangles, the heart sounds by the usual black bands.

The friction, as a rule, does not replace the normal heart sounds, but these, as well as loud endocardial murmurs, may be heard simultaneously with it. Their more distant quality tends to accentuate the superficiality of the friction sound. Moreover, the latter is considerably accentuated by moderate pressure of the stethoscope in the interspaces. As Emerson has shown, this is also true of certain endocardial sounds, but the latter are quite different in quality from a fresh pericarditis.

As the exudate is absorbed, the friction softens gradually into a barely distinguishable roughening of the first sound, and, finally, disappears altogether.

When portions of the exudate become organized and remain as thickenings of the endocardium, they may still give rise to some roughening of the first sound, which may be very difficult to interpret, and this is especially true when they remain in the form of fibrous strands and loose adhesions (see page 500).

The friction is usually first heard over the third and fourth left interspace near the sternum, over the area at which the exudate first appears. In the cases which are secondary to pneumonia and pleurisy there is often a "*pleuropericardial*" friction, with respiratory accentuation heard over a considerable strip along the left margin of the heart where

the pleura overlies the pericardium. The pleurisy exists in the layers of pleura in front of the pericardium. The two processes exist simultaneously in separate cavities whose walls are in contact. Sears has pointed out that in recurrent attacks of pericarditis the friction may be heard only at the back in the left interscapular region. This occurred in a case in which the anterior portion of the pericardium was adherent. The pulse in acute pericarditis is usually small and rapid, the blood-pressure low (100 mm. or under), and the pulse-pressure small (10–25 mm.), but this is, at least in part, due to the loss of vasodilator tone, and in part to the myocardial weakening brought about by the same process.

DIAGNOSIS.

Diagnosis is usually simple, and the murmurs are rarely mistaken for endocardial, although Osler mentions one case in which a to-and-fro aortic murmur was mistaken for a pericardial rub.

When a certain amount of fluid is present in the pericardium the friction may disappear if the heart is pushed backward; and, as a small pericardial effusion is often overlooked, the whole condition may escape diagnosis.

CASE OF SIMPLE FIBRINOUS PERICARDITIS.

F. G., a colored hod-carrier, aged 50, entered the Johns Hopkins Hospital on July 22, 1904, complaining of pain around the heart. Except for the fact that one son died of galloping consumption, the family history is negative.

The patient has been a healthy man, but had measles, chicken-pox, whooping-cough, mumps, scarlet fever as a boy, and rheumatic fever at 48. He is not subject to sore throat. He had a cough with pain in the chest twenty years before admission but has had no recurrence. He has had several attacks of gonorrhœa. He has always done hard work.

He was perfectly well until six weeks before admission, when he had *severe pain in the right thigh and hip* which lasted five weeks but he kept at work in spite of the pain. Four days before admission he began to cough, and two days later felt a cutting *pain around the heart*, which was especially severe on drawing a deep breath. This has persisted. He did not notice any special shortness of breath, and kept at work for three days after the precordial pain had set in.

The examination note by Dr. Cole states that the patient is a well nourished colored man, mucous membranes of fair color, no glandular enlargement. Lungs clear throughout on auscultation and percussion. Over the heart a feeble impulse is seen in the fifth left interspace 8.5 cm. from the midline. The impulse is localized. There is no bulging of the interspaces. On percussion the area of *cardiac dulness* is found to extend 11.5 cm. to the left of the midline in the fifth interspace, 3.0 cm. to the right opposite the fourth rib. The cardiohepatic angle is 90°.

The heart sounds are distinctly heard at the apex, but there is also a *loud rough pericardial friction* which is not exactly synchronous with the heart sounds and is increased by pressure with the stethoscope. There are no endocardial murmurs. The friction *increases in intensity toward the base* of the heart, where the heart sounds are distant and the second pulmonic is louder than the second aortic. The pulse is of fair volume, moderate tension, regular, 88 per minute.

The sputum is mucopurulent, but contains no tubercle bacilli and no elastic fibres.

Urine, 450 c.c., amber colored, acid, containing a considerable amount of albumin and numerous hyaline casts. Blood count shows: red blood-corpuscles 4,500,000, hæmoglobin 50 per cent.; leucocytes 7100. Temperature ranges from 101° to 102.5° F.

An ice-bag was kept continuously over the precordium, and he was given strychnine, 1.5 mg. (1/40 gr.) every four hours.

On the second day after admission the temperature fell to normal and the patient felt better. The pain in the chest had gone. The pericardial friction was still heard, but less intense than before, and by the following day could be heard only over a small area in the fourth left interspace near the sternal margin. It disappeared entirely during the course of the following week, and he became entirely well. There were no signs of pericardial adhesion. The patient left the hospital on August 8, in the third week after his admission, and has not sought admission since then.

TREATMENT.

Absolute rest in bed is necessary, since the heart must be spared as much as possible, and, moreover, cardiac strain and venous stasis tend to increase the exudation. The diet should be light or should consist of milk alone during the acute stages. If there is much pain, morphine may be freely given to relieve it, since this symptom is not likely to become chronic; and, on the other hand, it is important to keep the heart's action as quiet as possible. For this purpose an ice-bag is usually applied to the precordium.

Silva has shown in dogs that by this means the local temperature within the pericardium may be lowered 1° or even 3.5° C. (1.8°—7.3° F.). Buxbaum states that the use of the ice-bag in pericarditis is now quite general throughout the world. In American clinics this is certainly the case. The clinical experience of the writer has been one of uniform satisfaction in its use. The fact that Rubino was able to produce pericarditis in animals by the intravenous injection of cultures of pyogenic cocci only when ice was simultaneously applied to the chest, has probably little bearing upon the therapeutic use of the latter, since Rubino probably chilled his animals severely, while in the therapeutic application the cooling, especially of the deeper layers, is both localized and mild. No doubt the local temperature of the inflamed pericardium is reduced to normal, but not much below it.

Other methods of counterirritation are also useful and devoid of this possible objection. Head cites a case in which relief of the pain within five minutes was brought about by the application of three leeches to the precordium, and Biers's suction cups or the old-fashioned dry or wet cupping may be resorted to with equal satisfaction. This may also be said of hot or warm poultices, hot-water bags, and the modern electrical heating pads whose temperature can be kept regulated with great accuracy. Blisters (cantharides), mustard plasters, Paquelin cautery, and Finsen light may also be used with great satisfaction, or even a "light bath" from a single small incandescent lamp with reflector placed near the precordium.

Medicines seem to be of little value. Caton recommends potassium iodide. Broadbent states that digitalis should be avoided in the early stages, but Romberg recommends its use in the cases with nephritis. It should be used at once and should be preceded by intravenous strophanthin if signs of acute cardiac failure manifest themselves.

The pain is not relieved by salicylates, so that codeine, .03 Gm. (½ gr.), heroin, .005 Gm. (1/12 gr.), dionin, .02 Gm. (⅓ gr.), or morphine, .016 Gm. (¼ gr.), must often be given.

The bowels should be kept moving easily with saline purgatives.

PROGNOSIS.

Osler states that "simple fibrinous pericarditis never kills," but pericarditis is frequently seen as a terminal event in other conditions, especially in pneumonia, tuberculosis, gout, and nephritis. In any of these it is a grave but not always fatal sign.

The main dangers accompanying fibrinous pericarditis are the development of effusion on the one hand or of pericardial adhesions on the other. Sometimes all three conditions occur successively in the same case, the fluid collecting within a few days after the fibrinous exudate, is removed after a few weeks by aspiration or absorption, and is followed by organization of the exudate with adhesions which usually last throughout one or several years and finally terminate the life of the patient. Fortunately, these complications are by no means the rule, and in many cases fibrinous exudate is absorbed without further trouble.

It is evident from both the pathological and the clinical stand-point that the fresher the exudate the cleaner will be its absorption. Hence the importance of vigorous treatment.

PERICARDITIS WITH EFFUSION.

Frequently during the course, and especially in the second or third week, of an acute pericarditis fluid collects within the pericardium. Under normal conditions there are from twenty-five to fifty cubic centimetres of serous fluid present. In pericardial effusions from 500 c.c. to 1 litre is frequent; as much as 1000 c.c. has been found at autopsy by Verney. A pericardial membrane of the usual size could not accommodate so large an effusion, and stretching of the former usually goes on simultaneously with increase in the latter. Hence it follows that the actual size of the effusion may be of little import, and a small rapidly exuded effusion may produce signs of intrapericardial pressure sooner than a large one arising slowly.

The character of the fluid may vary as much as its quantity. It may be thin and serous and free from coagula, especially when poor in leucocytes, or it may contain small gelatinous coagula forming here and there about masses of leucocytes; or this process may be so generalized that the whole mass may be converted into a very thin jelly. Diapedesis of corpuscles through the injured vessel walls may cause it to become bloodstained, a condition which is especially frequent in carcinomatosis or sarcomatosis of the pericardium.

PATHOLOGICAL PHYSIOLOGY.

François-Franck, Lagrolet, and Cohnheim have shown that the injection of fluid into the pericardial cavity hinders the entrance of blood into the auricles, and thus causes stasis of blood in the venæ cavæ and fall in the blood-pressure. Their experiments were repeated by Starling, who found that on injecting successive amounts of 20 c.c. and 10 c.c. of oil into the dog's pericardium, the pressure in the vena cava rose gradually, while that in the aorta and pulmonary vein remained constant for some time. That is to say, the rise in venous pressure compensated for the

increased pressure within the pericardium, and although some venous stasis occurred the circulation was not retarded. As much as 60 c.c. of oil could thus be injected into the dog's pericardium without producing any other change. But when 10 c.c. more were injected the condition changed suddenly and completely. The small excess of fluid in the pericardial cavity had caused the pressure within it to rise considerably above that in the veins, and above the level to which the venous pressure could rise during stasis. The walls of veins and auricles, therefore, collapsed under excess of pressure (Fig. 281), and, since but little blood could enter the ventricles, the blood-

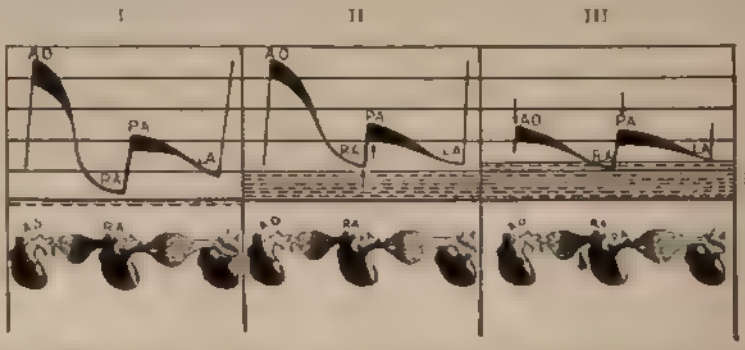

Fig. 281.—The circulation in cases with pericardial effusion. Diagrammatic. I. Normal. II. Small effusion, showing the rise in venous pressure and the increased difficulty in the filling of the heart. III. Great increase in intrapericardial pressure, showing the complete cutting off of venous inflow and fall in arterial pressure. The area shaded with horizontal broken lines indicates the height of the pressure within the pericardium.

pressure in the aorta fell. The same change occurred in François-Franck's experiments when the intrapericardial pressure was raised from 10 mm. to 20 mm. Hg. When the pressure was not relieved death ensued, the heart beating for a short time after the circulation had ceased. On the other hand, when the small excess of fluid was removed, the blood-pressure quickly rose again and the circulation returned to normal. This experiment exactly reproduces the condition in man when a pericardial effusion is collecting, illustrates the mechanism of death in that condition, and also illustrates the beneficial effect obtained from paracentesis when even a small amount of fluid is removed.

SYMPTOMS AND COURSE.

Pericardial effusion is somewhat less frequent in children than are the other forms of pericarditis, its subjects being usually adults and often persons past middle age. The symptoms of pericardial effusion are more insidious than those of the fresher fibrinous inflammation, pain being somewhat less common and less intense, dyspnœa and weakness being more intense. The patients are very much more comfortable in the vertical than in the horizontal position, the difference being even more striking than in the ordinary forms of heart disease. Few clinicians indeed can concur in James Mackenzie's statement that the presence of fluid in the

pericardium does not give rise to symptoms of circulatory embarrassment. Fainting spells and sudden death are very common, occurring when the inflow into the auricle is obstructed.

PHYSICAL SIGNS.

The patients are usually pale and weak with rapid respirations. The veins of the neck and extremities may be prominent (high venous pressure), and this is especially marked when the intrapericardial pressure is approaching the danger point. There may be inspiratory distention of the veins.

Inspection of the thorax usually shows a fulness of the interspaces over the precordium, and frequently a very diffuse wavy impulse which is lacking in the definiteness usually seen in both systolic impulse and systolic retraction over and about the normal or enlarged heart. Neither this nor the presence of a cardiac pulsation outside the apex is of real value in establishing the diagnosis of pericardial effusion.

Palpation, as a rule, reveals nothing of importance, except that the cardiac impulse is usually very feeble or absent.

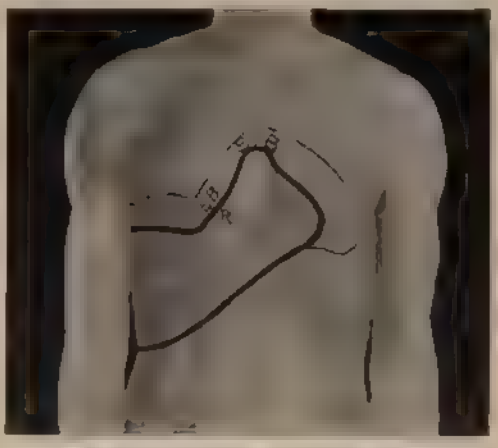

FIG. 282.—Area of cardiac dulness from pericardial effusion, showing Corvisart's triangular area of dulness and S-shaped posterior area of flatness. Rotch's area of dulness. *R* in the fifth right interspace. Ebstein's obtuse cardiohepatic angle. *EB*.

Changes in Cardiac Outline.—The pathognomonic sign is revealed by the alteration of dulness on percussion. Auenbrugger in his first diagnostic efforts was able to demonstrate a great increase in cardiac dulness in pericarditis with effusion, and this observation was confirmed by Corvisart, who recognized a large area of flatness in the form of a triangle with base downward. This, however, was also encountered in numerous cases of dilated heart and led to many errors in diagnosis, until T. M. Rotch, of Boston, in 1878, demonstrated that flatness was present in the fifth right interspace early in the disease and constituted an almost diagnostic feature. He was able to prove this upon the cadaver by injecting various quantities of cocoa butter into the pericardial cavity. Flatness in the fifth right interspace appeared whenever more than 200 c.c. of cocoa butter had been injected. Less than 200 c.c. could not be recognized.

Rotch's observations were confirmed by W. Ebstein in 1893, who laid stress upon the obtuseness of the angle formed by cardiac and liver dulness (cardiohepatic angle). This dulness is particularly marked when the patient leans forward and toward the right, so that the

fluid gravitates to this point. The right border of an enlarged heart, on the other hand, always forms an acute, or at most a right, angle with the liver dulness, and flatness rarely extends to the fifth right interspace, being most marked in the fourth. The matter has been still further investigated by Aporti and Figaroli, who found that with the subject in the vertical position as little as 150 c.c. of fluid showed itself by pushing the area of dulness downward and outward at both its lower angles—at both cardiohepatic angle and at the apex. The lower border of flatness is, therefore, the arch with concavity downwards which had already been described by Concato. When more fluid collects, the pericardium becomes more tense, all the surfaces become convex, and Concato's arch disappears. As a diagnostic sign the variations in dulness about the apex are much less definite than in the fifth right interspace, and hence they are of little importance.

At the upper border of dulness the usual slight resonance behind the sternum gives way to a tongue of absolute flatness when the exudate is large, so that the dulness assumes the form of a pear hanging from its stalk (Sibson).

Moreover, the pericardium presses upon the lungs about its borders, causes them to relax and give rise to Skodaic tympany and tubular breathing not only in front but also at the angle of the left scapula (Ewart.) Flatness may also be observed over the spines of the vertebrae, especially from the fifth to the tenth, where, as found by Koranyi, the note is normally resonant. This sign may also be present when the left auricle is greatly dilated, as in mitral insufficiency.

Position of the Heart in Pericardial Effusion.—The signs on auscultation may vary. Most commonly, as found by Pirogoff and subsequent writers, the heart sinks in the pericardial fluid and comes to lie against the vertebral column and away from the chest wall, from which it is separated by a thick layer of fluid. This fluid muffles the heart sounds, which may be totally absent, disappearing first about the apex, later at the base. Aporti and Figaroli have shown that with 650 c.c. of exudate a very small area of heart wall near the base will still remain free from fluid, and over this the heart sounds and friction rub may be heard.

On the other hand, the heart sounds and friction may persist even when a large amount of fluid is present, as in the case reported below, in which the pericardium contained 1200 c.c. of fluid. The sounds were faint at the apex, but became more distinct as the base was approached, where the friction was also well heard. An aspirating needle introduced in the sixth left interspace came at once against the heart. At autopsy the heart was found lying against the chest wall. This anterior position, though not the usual one, is, according to Schaposchnikoff, often assumed by the heart of a cadaver when fluid or paraffin is injected into the pericardium. Schaposchnikoff believes that the heart is held in this position in spite of the force of gravity by the elasticity of the great vessels.

Abdomen.—The liver may be both enlarged from the venous stasis and pushed down by the pericardial effusion, so that its lower edge is frequently palpable, sometimes even as low as the umbilicus. The spleen

may also be somewhat enlarged. Ascites and movable dulness are sometimes present.

Over the extremities the veins may appear distended, and there is often œdema.

Blood-pressure.—The blood-pressure is usually rather low, except in the cases with nephritis, in which it may be above normal.

The pulse is usually small, frequently collapsing, and often of the type of pulsus paradoxus—fall of blood-pressure during inspiration, with decrease in the size and frequency of the pulse, and, on the other hand, inspiratory swelling of the veins. This condition is due to traction on the walls of the vena cava producing stasis during inspiration.

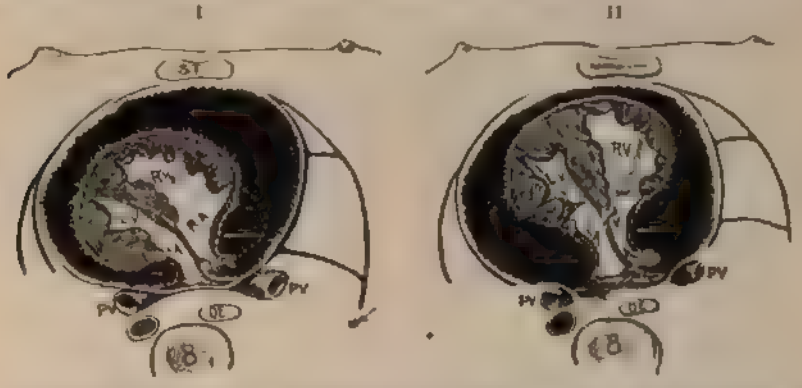

Fig. 283.—Positions of the heart in pericarditis with effusion. I, The fluid is in front of the heart, as described by Pitogoff. II, The heart is floated up against the chest wall, as described by Schuposchnikoff. *ST*, sternum; *OE*, œsophagus; *PV*, pulmonary veins; 8, eighth thoracic vertebra. The arrows indicate the compression of the auricles. The broken line indicates the outline of the uncollapsed auricle.

X-ray Examination.—Examination with the fluoroscope shows the exact size, form, and position of the effusion (Fig. 283), and both before and after paracentesis may be of great help in locating pockets of encapsulated fluid. The relations to the diaphragm and the presence of mediastinal adhesions may sometimes be diagnosed by this means, and particularly by means of permanent radiographs made with a tube of low vacuum.

Case of Pericarditis with Effusion

The following typical case was under the writer's care in the Johns Hopkins Hospital. It has been previously reported in considerable detail by Professor Thayer.

R. C. W. B., a German saloon-keeper, aged 59, was brought to the hospital at 12.30 P.M. on Sept. 1, 1903, barely able to speak, owing to *shortness of breath and weakness*. He was too ill to give a history, except for the statement that for several years he had been troubled with shortness of breath, which has gradually increased until the past few days, when it suddenly became very much aggravated.

Examination note by Dr. Cole was as follows: Patient is a moderately well-nourished man; muscles flabby. At time of the examination he is lying flat with head slightly propped up and looks very ill. Respiration 40 per minute. Pupils are small, react readily to light. Tongue dry, slightly coated. No marked pyorrhœa; teeth not good. *Veins* of the neck are very *full*; no marked pulsation of the deeper vessels. No general glandular enlargement. *Chest*: Expansion fairly good, equal, considerable respiratory distress. Resonant throughout right front and axilla and left upper front, but note is markedly impaired in lower left axilla and at lower right back, much more markedly in lower left

back up to the angle of the scapula. On auscultation.—Left: Breath sounds are clear throughout upper front and upper back except for a few mucous râles in interscapular space. Below they are very distant, practically absent at the extreme base, except just at the angle of the scapula, where they are a little harsher (Ewart's sign). Right side: Breath sounds are quite clear throughout except in the lower back where there are mucous râles and breath sounds are distant.

Heart.—There is no impulse visible or palpable. There is a wide area of cardiac dulness extending above to the middle of the third rib 16.5 cm. to the left of the midline in the fifth interspace when the patient lies on his right side. When on his back, however, the dulness is difficult to make out as it extends directly to the dulness in the axilla. On the right dulness extends apparently 7 cm. to the right of the midline in the fourth interspace. The angle between the upper limit of liver dulness and the cardiac dulness is very obtuse. There is quite definite precordial bulging, though the intercostal spaces seem no fuller than on the right. At the apex and over the entire precordium the heart sounds are barely audible until one reaches almost to the costal margin in the fourth and fifth left interspace, where the sounds are heard faintly with a to-and-fro murmur, which is also heard over the sternum from the third to the fifth rib. This murmur is quite superficial and is louder during expiration than during inspiration, apparently not increased by pressure of the stethoscope. It sounds suspiciously pericardial in character, but not definitely so. The heart's action is almost fetal in rhythm (pulse-rate 138 per minute). Heart sounds are heard more loudly in the second left interspace; neither aortic nor pulmonic second is specially accentuated. Pulse is very small and can hardly be counted.

FIG. 284.—Radiograph of a patient with pericardial effusion, taken with the tube in front of the patient. Kindness of Prof. I. M. Cooper. The figure shows the overflowing of the pericardial exudate. The spots over the surface of the lungs are artefacts due to the presence of air-bubbles in the developer.

The abdomen is full. There is no movable dulness in the flanks. The feet and legs are markedly œdematous.

At 5.45 P.M. the patient was prepared for paracentesis pericardii by the method of Delorme and Mignon under aseptic precautions. An incision was made in the fifth left interspace at the sternal margin, the pericardium exposed and a trocar inserted through it. By means of a Potain aspirator 25.50 c.c. of thick serosanguineous fluid were removed, after which no more could be removed. Another puncture was immediately made in the costoxiphoid angle and a small amount of fluid again removed. The patient's pulse and general condition did not change. He became delirious and died at 7.30 P.M.

The autopsy findings confirmed the clinical observations. There were however, still 1200 c.c. of pericardial fluid which lay behind the heart both to the left and to the right. The heart lay directly against the chest wall, and therefore had come against the point of the needle and prevented the removal of the fluid. As Dr. Cole remarked in a subsequent note: 'the fact that the heart sounds were heard loudest over the sternum and along the left sternal margin should have led me to insert the needle either to the right of the sternum or far to the left—outside the mammillary line'—preferably the latter. The pericardium was lined with a yellow fibrinous exudate. The heart muscle showed cardiosclerosis. The coronary

arteries were tortuous. There were thickening of the aortic valves and adhesions of the cusps, which gave rise to slight aortic stenosis. The left pleura contained 700 c.c. of slightly turbid straw-colored fluid. Both lungs contained small areas of tuberculous bronchopneumonia.

PURULENT PERICARDITIS.

The effusion in many cases is purulent and associated with more or less severe septic symptoms, septicæmia, chills, extreme pallor and weakness, and, as a rule, a septic fever, though in some cases the temperature remains normal.

The condition may follow exposure to bad weather, empyema, or trauma to either the front, sides, or back of the chest. The staphylococci, streptococci, pneumococci, gonococci, and a great variety of other bacteria may be the infective agents.

The symptoms and physical signs are very similar in both simple and purulent effusions. The history of trauma and the presence of empyema or other foci of pus speak in favor of a purulent effusion. Leucocytosis may be present in both conditions. The aspirating syringe usually gives the diagnosis.

HYDROPERICARDIUM.

Hydropericardium, or simple serous effusion into the pericardium, may occur along with ascites, hydrothorax, and general anasarca in chronic heart failure or in nephritis, or it may occur alone as a result of local venous stasis from the pressure of mediastinal growths, glands, or aneurisms, or from strangulation of the veins by adhesions. In this case an afebrile course is run. Leucocytosis is often absent (or may be due to simultaneous bronchitis or bronchopneumonia), and the diagnosis rests upon the signs of the intrathoracic condition which is the causal factor.

The differential diagnosis is made from the fluid obtained on paracentesis, which is clear, thin, serous, of low specific gravity (under 1018), and gives no clouding with acetic acid indicative of nucleo-albumin. It is poor in leucocytes and fibrin ferment, and there is not much albumin (shown by Esbach's method). The presence of a pericardial friction at any time during the course of the disease is sufficient to exclude a simple hydropericardium.

HÆMOPERICARDIUM.

Hæmopericardium is produced by the effusion of pure or almost pure blood into the pericardium, and occurs especially as a result of direct or indirect trauma, stab or gunshot wounds penetrating the cavity, or rupture of the heart or of an aneurism. It may also occur from erosion of a blood-vessel by a malignant growth.

Hemorrhage into the pericardium takes place much more rapidly than the other exudations, so that the pericardium has less opportunity to stretch and accommodate itself to its contents. The intrapericardial pressure, therefore, rises more rapidly than in the other conditions, and symptoms, signs, and danger develop more rapidly. Death may occur at once. When possible operative procedures must be begun promptly in order to save the patient.

PNEUMOPERICARDIUM.

When air or gas enters or develops within the pericardial cavity (as from perforation after trauma or tuberculosis or infection with Bacillus aerogenes capsulatus Welchii in a case reported by Nicholls), the condition is termed pneumopericardium. Usually this is associated with the presence of purulent or serous fluid (pyopneumopericardium, pneumohydropericardium). Since there is normally a negative pressure (—3 to —5 mm. Hg) within the cavity, it follows that air will enter, just as into the thorax (pneumothorax), whenever there is a free perforation to the outside or to the air-passages. This is most frequent in perforating wounds, but occasionally occurs as the result of tuberculosis or perforation of a purulent pericarditis.

The signs of pneumopericarditis are very characteristic. The percussion note over the cardiac area may vary from a bell-like tympany to an absolutely wooden flatness, or when there is an opening of medium size a cracked-pot note may be heard. With a free communication to the outside such as results from operation upon the pericardium, however, the air within the pericardium is not set into vibration by the percussion stroke and does not alter the note at all.

On auscultation, except in the latter condition, a loud churning "mill-wheel" murmur is heard, but when the communication is a free one this may be totally absent.

TUBERCULOUS PERICARDITIS.

Tuberculous pericarditis is a common and severe condition. The fibrinous stage is somewhat more chronic than in the other forms of pericarditis, lasting several weeks or months, and often resulting in the formation of deposits of fibrin (Fig. 256) an inch in thickness, with or without the presence of fluid. Gray tubercles of various size may be visible within and upon the surface of the exudate, but frequently they may not be present, and the bacilli must be sought for histologically or by guinea-pig inoculation.

The fluid in pericardial effusion is frequently blood stained. It is occasionally purulent (Kast). Tuberculous pericarditis is most commonly associated with other tuberculous processes, especially involvement of the pleurae, but it may also occur as a "primary" manifestation by spreading from caseous mediastinal lymph-glands.

The course, though more chronic than other pericardial processes, is quite similar, but the exudate is not absorbed completely and goes on to either fluid or adhesive pericarditis. Frequently both conditions occur and encapsulated effusions result. There is usually a considerable rise of afternoon temperature.

The physical signs and therapy of tuberculous pericarditis are about the same as in the other forms, plus the general management of a case of tuberculosis—rest, fresh air, very liberal diet (when cardiac symptoms have subsided), and sustaining measures. The prognosis is bad.

TREATMENT OF PERICARDITIS WITH EFFUSION.

Palliative treatment of fluid within the pericardium must be limited to the periods in which intrapericardial pressure is well below the range of venous pressure, and must be pursued with full cognizance of the fact that death may ensue whenever the pressure exceeds this limit.

The palliative measures consist of counterirritation, with ice-bag or poultices, etc., blisters, and especially application of Bier's suction cups or leeches. Diuretics,—theocin, diuretin,—combined with digitalis or strophanthus, and free purgation may be resorted to, and the liquid intake restricted to below 1000 c.c. per day, in the hope of reducing the pericardial fluid by these means. However, these methods are at best but feeble palliatives, and often more risk is entailed in their use than in the more radical procedures.

Paracentesis Pericardii.—The idea of removing fluid within by tapping the pericardium was first suggested in 1646 by Riolan, who advised trephining the sternum one inch above the xiphoid.[1] He did not attempt, however, to carry it out, and the first operation upon the pericardium was performed in 1819 by Romero, of Barcelona. Romero operated upon three cases of pericarditis, with two recoveries, a percentage which is above the average even for the present day.[2]

Puncture of the pericardium by means of a trocar was first performed by Jowett, of Nottingham, in 1827. It was brought into more general repute by Schuh, of Vienna, under Skoda's direction (1839), in France by Trousseau (1854) and by Aran (1855), and in England by Clifford Allbutt (1866). Paracentesis pericardii should, of course, be undertaken with all possible asepsis of skin, hands, and instruments.

The instrument used has varied from a thick trocar several millimetres in diameter to the finest aspirating needle. The ideal cannula is one which has a bore (about 1 mm.) sufficient to allow a viscous liquid to escape easily, and yet not so great as to permit the entrance of air through the perforation. A trocar and cannula, especially one ending in a T and stop-cock, is the best form of apparatus, since it permits the operator to clear the lumen of the cannula at will and at the same time to remove the fluid by suction through an aspirating bottle.[3]

Various sites for the paracentesis are recommended, with four ends in view:

1. To obtain the fluid.
2. To avoid infecting the pleural cavity and puncturing the lungs.
3. To avoid puncturing the heart.
4. To avoid injuring the internal mammary artery.

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[1] "Si non possis exhaurire istud serum per hydragoga, licet ne terebra sternum aperire, intervallo pollicis a cartilagine xiphoide."

[2] It is interesting that this method has recently been advocated by J. H. Bacon (A Procedure for Opening the Pericardium, Am. J. M. Sc., Phila. and N. York, 1905, cxxx, 652) as a result of a series of experiments upon the cadaver. Bacon does not mention the work of these pioneer surgeons.

[3] Dr. Chas. S. Bond has found a curved aspirating needle with lumen about 1 mm. in diameter very useful in tapping the pericardium when the fluid lies back or is encapsulated. The needle which he uses has a radius of about 10 cm. following the curve of the heart and enabling him to pass around the latter without injuring it. The danger of entering the ventricle by a straight push is also much less with an instrument of this form.

Sites for Paracentesis.—Trousseau (1854) recommended introducing the needle in the fourth interspace just below the mammilla; Dieulafoy (1873) in the fifth about six centimetres from the sternal margin. Puncture at these sites or at the outer border of absolute dulness (flatness) has the disadvantage of always traversing and often infecting the pleural cavity, so that occasionally the patient may be caused gratuitous empyema or even a fatal pneumonia.

In order to avoid entering the pleural cavity, Baizeau (1868) and Delorme and Mignon advocate puncturing the pericardium as near as possible to the sternal margin in the fifth, or if possible the sixth, left interspace. In order to render the procedure more certain, the latter investigators advise making an incision through the skin with a bistoury. The needle (of medium diameter) is then introduced into the sixth interspace if possible, and otherwise into the fifth along the edge of the sternum, pushed in for a centimetre or two, and then the point directed downward and inward by a slow continuous movement until the liquid emerges. In order to empty the pericardial cavity the needle should be connected with an aspirator bottle and the fluid collected by gentle aspiration.[1] When the instrument is inserted slowly in the manner described, the risk of injuring the heart (right ventricle) is minimal, for the beating of the latter against the point can be felt as soon as it is touched and long before it can be penetrated. Even if through lack of care the right ventricle be penetrated, harm rarely results. For example, Hulke mentions a case in which he penetrated the right ventricle and a few jets of blood spurted out, but the patient's condition improved; and he cites several other similar cases. Only one case of death (from laceration of the right ventricle due to paracentesis is on record (West). Unquestionably when all goes well the technic of Delorme and Mignon is the most satisfactory, since the danger of injuring both heart and pleura is minimal. On the other hand, the chance of a 'dry puncture' is great. At the place selected the point of the needle may penetrate a great deal of dense fibrous tissue and even periosteum and the lumen may thus become plugged. Should the fluid not appear, this source of error may be obviated by carefully inserting a wire through the whole length of the needle after it has been pushed into the cavity and then withdrawing the wire. Another difficulty may lie in the position of the heart itself, as occurred in the above-mentioned case of the writer's, in which the heart instead of lying behind the fluid lay directly against the chest wall in the position described by Schaposchnikoff. When the needle was introduced by Dr. Cole, it encountered the heart at once, and the rubbing of the latter against the point could be readily felt. This might have been prophesied from the fact that the heart sounds were well heard over the precordium. With the exception of a few cubic centimetres of clear fluid the puncture was a dry one, in spite of several successive insertions of the needle both at this point and in the costoxiphoid angle. The patient's condition became very bad, and he died before a second paracentesis could be undertaken. Autopsy showed the heart lying directly against the chest wall with 1200 c.c. of fluid above and to the left. In this case, as in all those in which the heart sounds and pericardial friction are well heard at the time of paracentesis,

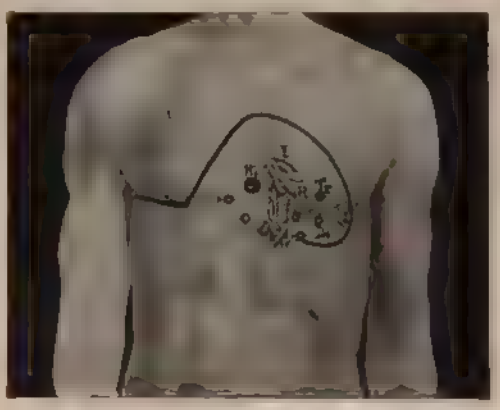

FIG. 285.—Sites for paracentesis pericardii and pericardiotomy. *R*, Roslan (1840), trephining the sternum. *D & M*, Delorme and Mignon (1895), paracentesis. *R*, Romero (1819), pericardiotomy. *E*, v. Eiselsberg's pericardiectomy. *T*, Trousseau (1854). *Di*, Dieulafoy (1873), paracentesis. *W*, West, pericardiotomy (1883).

[1] Sewall, J. Am. M. Asso., Chicago, 1900, advises aspirating the fluid into the aspirator bottle by sucking out the air with the mouth instead of with a mechanical aspirator. The procedure is simpler and mistakes and failures of the pump are impossible.

it would have been better to have introduced the needle at the outer border of cardiac flatness in spite of puncturing the pleura, and to have risked empyema to save the patient.

Drainage of the Pericardium.—Prof. Pearson, of Cork, punctures the pericardium with a large trocar, withdraws, and then introduces a fine rubber catheter into the pericardial cavity through the tube of the trocar. The rubber catheter follows the curves of the pericardium without danger of rupturing it, and thus enables him to reach exudates which, as in the case of R. C. W. B. cited above, are located behind the heart. He also withdraws the metal tube and leaves the rubber tube in place as a permanent drain for several days at a time, and states that in this way he has been able to cure a number of stubborn cases of chronic pericarditis with effusion which had resisted all other methods of treatment.

The fact cannot be too greatly emphasized that cases with pericardial effusion are usually desperate cases, and the fluid should be gotten out at all hazards. It is true that all the fluid need not be removed to effect recovery, since the removal of a small amount, just as in Starling's experiment, allows the circulation to re-establish itself and often permits the rest to be absorbed.

Resection.—As has been seen, paracentesis pericardii, even in cases of simple serous pericarditis, may be far from satisfactory. In purulent pericarditis and hæmo- and pneumopericardium it is still less so. In such cases paracentesis is inadequate and the pericardium must be opened freely. Radical as this procedure may seem, its satisfactory performance by Romero antedates paracentesis. Romero made an incision in the fifth intercostal space at the level of the costochondral articulation, introduced his finger into the wound, palpated the pericardium with his finger, and then seized it with forceps and opened it with curved scissors. The operation is best performed under light chloroform anæsthesia. Though this must be carefully administered on account of the cardiac weakness, it is a significant fact that most of the patients have stood the anæsthetic well.

The site for free incision has varied with different operators. Rosenstein made a free incision in the fourth left interspace close to the sternum and then inserted a rubber-tube drain. West operated in the fifth left interspace in the nipple line, having previously introduced an aspirating needle, which he used as director for a long narrow-bladed sharp-pointed bistoury, subsequently enlarging the opening with a probe-pointed bistoury. V. Eiselberg resected the fourth costal cartilage and then opened the pericardium. Delorme and Mignon perform what is probably the least dangerous and most satisfactory operation. They disarticulate the fifth and sixth costal cartilages from the sternum with a pointed bistoury, draw them forward one by one, and fracture them about 4 cm. from the sternum. They then dissect down to the pericardium, which they pull forward with forceps, and then slit it up with scissors for several centimetres.

Many observers, from Aran to the present, supplement the simple drainage with irrigation of the pericardium. Aran injected a dilute tincture of iodine at 100°, a procedure which in his case (though not in all others) did not cause pain. West used warm 1 per cent. carbolic acid; others used simple salt solution. The importance of irrigation cannot be too freely emphasized, since the treatment should aim not only at recovery but also at reducing the exudate and the resulting adhesions to a minimum.

Delorme and Mignon operated upon all forms of pericardial effusions. Their conclusions are summed up in the following: "100 observations

—82 paracentesis, 18 incision: 82 paracentesis—mortality 65 per cent; 18 incisions—mortality 38 per cent. Let us do for the pericardium what we have done for the peritoneum."

The relative merits of palliative therapy, paracentesis, and free incision are well shown in West's case of purulent pericarditis:

A van boy, aged 16, was struck in the back by a truck and knocked down. No symptoms for two months, then shivering and pain in the left side and precordium. Pain subsided in a few days. Three weeks later he went out for a short walk; became very faint and almost fell down. Pain seized him in the pit of the stomach. Became cyanotic, dyspnœic, and nauseated. Admitted Sept. 7. Pulse 78; paradoxic, losing 2-3 beats at each inspiration. Precordial bulging and œdema. Dulness from right nipple line to three inches outside left nipple line. Cardiac sounds almost inaudible. Liver pushed down and felt in epigastrium. Slight œdema of feet.

Twelve leeches applied to the precordium followed by poultices. Palliative treatment for a week. Pulse and general condition feebler.

Sept. 14. Paracentesis pericardii—fourth left interspace below nipple; 90 c.c. 1 per cent. carbolic acid at 100° then introduced through the needle and used to wash out pericardial cavity. No pain. Patient much relieved.

Sept. 17. Patient's condition again bad. Paracentesis fails to remove fluid. Free incision under chloroform, as above described, in fifth left interspace, at least two quarts of pus removed. Immediate improvement. Uneventful recovery.

Left hospital Feb. 23, and the following September was perfectly well and had been following his usual work for the past six months as well as ever.

Rosenstein's case and those of Delorme and Mignon show similar results.

West gives the following statistics for paracentesis:

	Number	Recovery	Death.
Phthisis. .	13	4	9
Rheumatic fever	11	7	4
Scurvy	9	6	3
Pleurisy....	6	5	1
Injury	3	2	1
Pneumonia	2	.	2
General dropsy			
Morbus cordis	2		2
Nephritis.....	2	2	
Chronic bronchitis	1	1	
Mediastinal tumor	1		1
Unassigned..	17	7	10
	67	34	33

In spite of the comparative harmlessness and brilliant results obtained by the radical operation in purulent pericarditis, it is not probable that this procedure can be extended to the milder exudates, since, just as in joints, free prolonged drainage is followed by complete obliteration of the cavity. Irrigation of the cavity through an aspirating needle or trocar, after tapping, is possible only when the diameter is large and the outflow is a free one.

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ADHERENT PERICARDIUM

(Adherent pericardium, adhesive pericarditis, synechiæ pericardii, concretio pericardii cum corde, chronic mediastinopericarditis.)

Whenever a pericardial exudate, fibrinous or fluid, is absorbed slowly a certain amount of organization takes place in it and adhesions form just as after pleurisy or peritonitis. The form of these adhesions varies considerably, from long thin strands stretching like cords across the pericardial cavity to short bands of dense fibrous tissue, or even to a firm tissue which

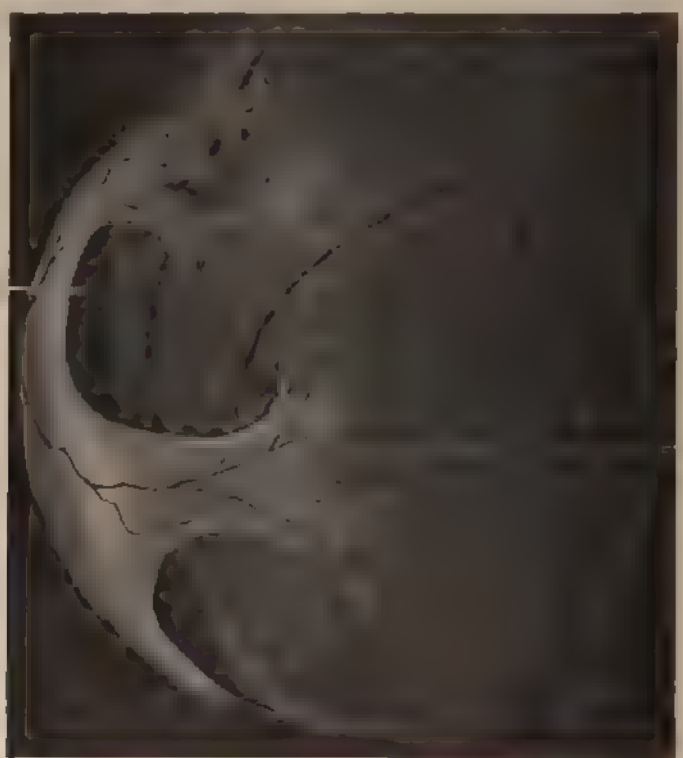

FIG. 280.—Specimen showing the two layers of pericardium united in some parts by long strands and elsewhere by short bands of dense adhesions. (From a specimen in the Army Medical Museum, Washington, D. C.)

knits the two surfaces together and completely obliterates the cavity. All these forms may be present in different areas of the same pericardium, so that the process need not be considered as perfectly homogeneous.

Moreover, not only the adhesions within the pericardium, but particularly the extrapericardial adhesions which are formed simultaneously on the outer surface are of clinical importance, since it is the latter which form the tightest lines in the harness and determine the strain upon the heart. As shown by Manges' case cited below, complete obliteration of the pericardial cavity may cause no symptoms as long as the extrapericardial adhesions remain unimportant.

The main adhesions do not always occupy the same position, but may be divided into the following groups (Fig. 288):

1. Chondropericardial—fixing the heart to the costal cartilages and chest wall in front.
2. Pleuropericardial—gluing it to the pleurae and fixing the edges of the lungs.
3. Mediastinopericardial—fixing its posterior surface and especially harnessing the auricles.
4. Phrenopericardial—fixing it to the diaphragm.

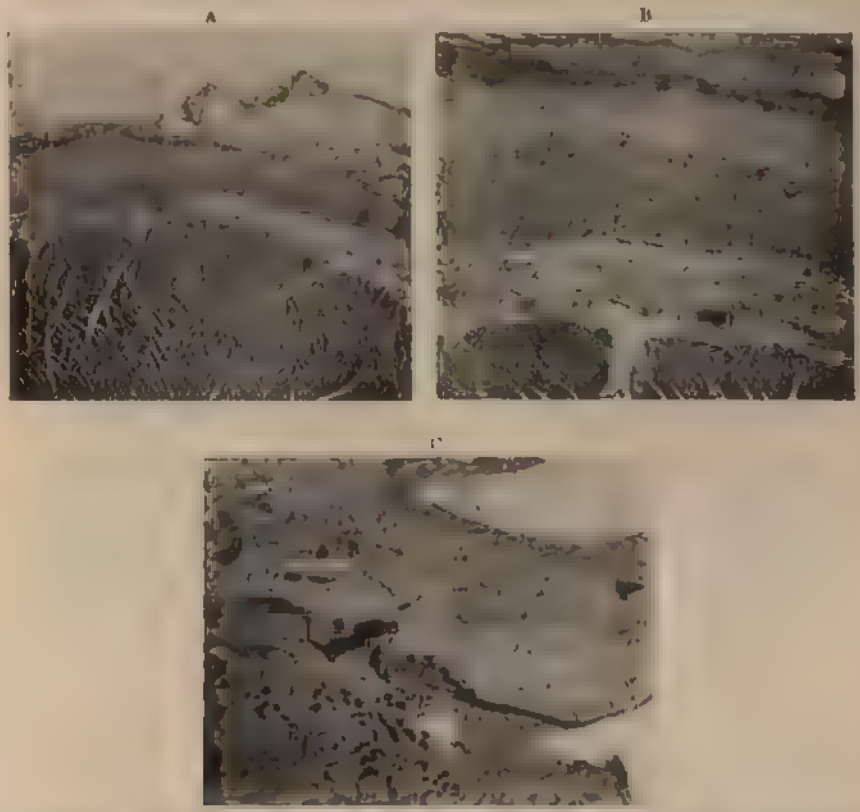

Fig. 287.—Sections showing universal pericarditis. Photomicrographs by Dr. C. S. Bond. A Seen with low power. B Same spot as under high power. C Another specimen showing the extreme vascularity of pericardial adhesions.

Each of these gives rise to a distinct group of physical signs; and, since these may occur separately, it is important that they should be considered so.

PATHOLOGICAL PHYSIOLOGY.

The mechanical effects upon the circulation due to pericardial adhesions may be twofold. 1. the work of the ventricle is increased by the tug upon the adhesions; 2. the filling of the heart may be hindered by strangulation of the vena cava. At each contraction the heart must not only drive out the blood, but must pull on its harness of adhesions. The additional work which it thus has to perform depends both upon the tightness of the

adhesions and upon the weight or rigidity of the structures pulled. The latter factor depends upon the position of the adhesions, whether it is the ribs, pleura, mediastinum, or the diaphragm and liver that are tugged upon, being greatest for adhesions to the ribs and diaphragm. 3. The emptying of the heart and the flow through the aorta may, as claimed by Kussmaul, be hindered by the tugging of the adhesions upon the arch of the aorta. This can readily be shown experimentally if such traction be made in a dog whose chest has been opened. The pulse may be made to disappear absolutely in spite of the fact that the heart rate remains unchanged and the heart dilates from overfilling; enough blood flows in from the vena cavæ to dilate the heart.

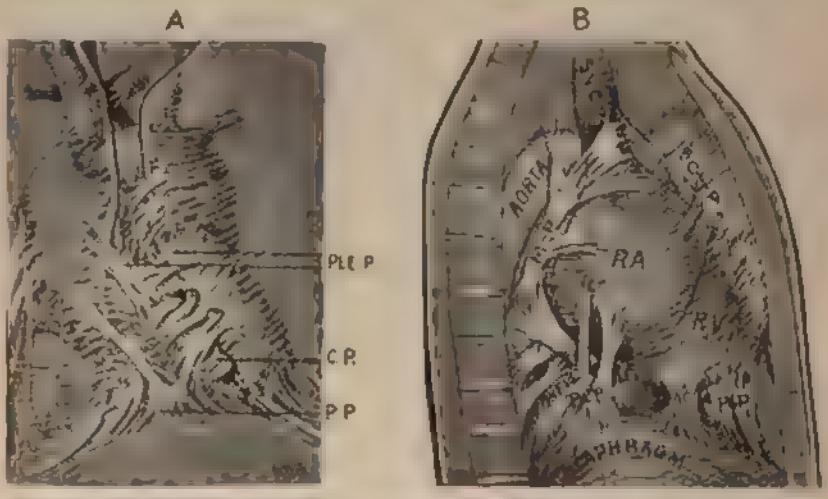

Fig. 288.—Anterior and posterior pericardial adhesions. (Semi-schematic.) A, Anterior adhesions showing the stumps of adhesions to the ribs. B, Mediastinal adhesions showing a side view of the heart. *Pl E P*, pleuro pericardial adhesions; *C. P.*, costo- (or chondro)-pericardial; *P. P.*, phreno-pericardial; *M. P.*, mediastino-pericardial adhesions.

When this additional work is imposed upon a heart already weak, it may succumb to the strain, and death may occur with all the manifestations of broken compensation. The importance of adherent pericardium in causing death from heart disease is shown by the fact that it was present in almost all the cases of Sturges' series.

Usually, however, the ventricles gradually recover from the strain and simply undergo a gradual work hypertrophy proportional to the additional strain, and an additional amount of work may be done at each systole sufficient to balance the amount required. During exercise, emotion, disease, or other strains, however, not only the work of the heart in the circulation is increased, but with the increased systolic output and systolic excursion of the walls the tug upon the adhesions is increased enormously, and the heart is thus readily overstrained. The heavy beating of the heart under emotional excitement is especially likely to bring this about.

Moreover, the process of hypertrophy is not a pure one. With the fibrosis of the pericardial adhesions outward, the process of fibrosis also

extends inward into the somewhat injured myocardium, and this process goes on progressively with each moment of overstrain until the myofibrosis cordis is advanced and the heart failure complete.

The site of the adhesions determines not only the degree but the character of the heart failure. If the densest adhesions are over the left ventricle, the effect is to inhibit the action of the latter alone. Nature performs the experiment of Welch, and gives rise to the clinical picture of broken pulmonary compensation with dyspnœa, cardiac asthma, or pulmonary œdema.

If the chief adhesions are over the right ventricle, on the other hand, broken systemic compensation sets in with venous stasis, tricuspid insufficiency, enlargement of the liver, and collection of fluid at various sites, but particularly in the peritoneal cavity (cf. Pseudocirrhosis, page 509).

On the other hand, the tugs of the adhesions on auricles and ventricles may act as mechanical extrastimuli and produce an extrasystolic arrhythmia, which in itself hinders the circulation.

SYMPTOMS.

Since the actual formation of the adhesions really represents the subsidence of the acute pericardial process, it is not surprising that the onset of the pathological lesion is insidious, and indeed may coincide with the subsidence rather than the onset of symptoms. This is well illustrated by cases of purulent pericarditis like that reported by Manges, in which obliteration of the pericardial cavity accompanied the curve of the healing of the incision. The patient was free from symptoms, and a year later was working as a messenger boy. In most cases the process continues insidiously during months or years before cardiac symptoms and heart failure set in, during which the patient may be apparently well or may suffer only upon over-exertion, over-indulgence in venere et potu, or emotional excitement. Sooner or later the pump wears out and symptoms become marked.

The symptoms of adherent pericardium are mainly those of chronic heart failure—palpitation, weakness, etc. Precordial pain localized about the apex or the base of the sternum is common (65 per cent. 70 per cent. of cases). As stated above, the other symptoms may fall into the category of cardiac dyspnœa or that of venous stasis and dropsy, dependent upon whether the failure of compensation is in the pulmonary or systemic circulation. In the former case there are attacks of coughing and acute dyspnœa, sometimes with smothering sensations. The latter often begins insidiously with weakness, enlargement of the liver and spleen, swelling of the abdomen (Pick's pericarditic pseudocirrhosis of the liver, or pericarditic polyserositis), and swelling of the feet. These symptoms may also set in more acutely as in the form of simple heart failure.

Delirium occasionally occurs with adherent pericardium, perhaps due to disturbed cerebral circulation. In one case under the writer's care the patient was subject to hallucinations of vision during the periods when his cardiac condition was bad. These were probably due to congestion of the retinal capillaries, so that he saw lions and tigers jumping over one another at the foot of his bed, even though he realized it was a physiological hallucination.

PHYSICAL SIGNS.

Corresponding to the variations in the site of adhesions, the physical signs of adherent pericardium are both multifarious and interesting. The patients are often pale and pasty, the haemoglobin being low and the capillaries rather empty of blood. Sometimes the opposite holds true, and plethoric cyanosis prevails. Inspection of the veins of the neck may show filling of the latter during inspiration (Kussmaul's sign), accompanied by inspiratory diminution in the size of the pulse or even omission of some beats during inspiration (pulsus paradoxus, Kussmaul) (see page 506). The sounds over the heart during this period may become weaker, but usually still continue.

The so-called Friedreich's sign (diastolic collapse of the vein), now known to represent merely a weak positive venous pulse (see page 57), is common to many weak hearts and has no diagnostic or prognostic value.

C. M. Cooper has recently added what seems to be a valuable accessory sign of adherent pericardium. He determines how long the patient can hold the breath in inspiration, and, five minutes later, the same for holding the breath in expiration. In normal individuals $\frac{\text{insp.} = 40\text{–}70}{\text{exp.} = 20\text{–}25}$; in cardiac lesion $\frac{25}{15}$, in persons with mediastinal and pericardial adhesions $\frac{\text{insp.} = 9}{\text{exp.} = 25}$ (paradoxical ratio). Patients with bronchial asthma also showed $\frac{\text{insp.} = 15\text{–}20}{\text{exp.} = 25\text{–}35}$ (paradoxical ratio), so that its chief value is as confirmatory evidence. The presence of a paradoxical ratio may prove very useful in confirming, and a normal ratio in excluding, mediastinopericarditis.

Broadbent's Sign.—The chest usually shows marked precordial bulging, especially in children. Walter Broadbent in 1895 called attention to a "visible retraction, synchronous with the cardiac systole, of the left back in the region of the eleventh and twelfth ribs," and "in less degree of the same region of the right back" (Broadbent's sign). Such retractions of the interspaces have also been recognized in many cases of cardiac hypertrophy by the Broadbents as well as by other observers (Tallant). J. H. F. Broadbent has lately (Heart Diseases, 4th edition) stated the facts more definitely and more accurately in the following words: "The systolic recession of spaces alone is, however, not a trustworthy indication, as it may be due to atmospheric pressure, especially when the heart is much hypertrophied. When the costal cartilages or lower end of the sternum are dragged in, there can be little doubt as to the diagnosis, as this could not be effected by atmospheric pressure." This sign is often most marked in deep inspiration when the diaphragm is tense.

Broadbent also states that systolic retraction over the apex is a valuable sign, but only when the impulse is forcible on palpation, as it may otherwise be due to atmospheric pressure (over the right ventricle; cf. page 91). This is certainly true in many cases, but in the writer's experience there are frequent exceptions to this rule, and it is of value chiefly as a corroborating sign.

Percussion.—The area of cardiac dulness is usually but by no means always enlarged, owing to the hypertrophy which usually takes place,

though fixation of the lung borders may cause the area of flatness and area of dulness on the left to almost coincide. The characteristic features on percussion are: Absence of the usual change in the left border of flatness between deep inspiration and deep expiration. This movement of the border of the lungs, which is normally 2-3 cm., may be reduced to less than 1 cm. or may absolutely disappear. The position of the apex, as determined by palpation, auscultation, and percussion, also becomes fixed, and may not change at all when the patient turns from lying on his right side to lying on his left. However, both these fixations may be present with simple pleural adhesions and no actual involvement of the pericardial cavity. This was well exemplified in the case of a little girl who had been a patient in the Johns Hopkins Hospital several times during the last couple of years, and who presented signs interpreted as adherent pericardium. At autopsy the pericardial cavity was free from inflammatory processes, but the pleuræ were everywhere bound down tightly around it. Practically the effects were nearly the same as if the pericardial cavity had been involved, Broadbent's sign and pulsus paradoxus being present to a slight degree. Such cases are, however, extremely rare, and difficult to diagnose when they occur.

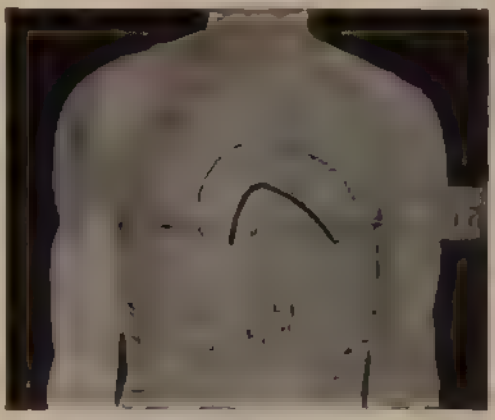

FIG. 289.—Cardiac outline in adherent pericardium. The broken line indicates the fixation of the left border of the heart, apex, and of the left border of cardiac flatness, anterior margin of the left lung. The small diagram at the left shows the relation of the heart sounds to the cardiac cycle, indicating the unusually loud third heart sound. *BR-BR* indicate areas of systolic retraction of the ribs, xiphoid, and interspaces. *R* 1, 2, Riess' gastric sounds in adherent pericardium.

Palpation. — Sir William Broadbent has called attention to the importance of an exaggeration of the diastolic shock or rebound (accompanying the second sound) over the greater part of the pericardium as characteristic of adherent pericardium. This is certainly a useful aid especially in corroboration of other signs, but, unless the distinctness of the shock is far greater than would be warranted by the loudness of the sound at the base, it is of little value. Nevertheless, the writer recalls a case in which the diagnosis of adherent pericardium (accompanying a well-defined aneurism) was based upon this sign alone and was verified at autopsy. Professor Thayer has found that there is often in addition a protodiastolic shock accompanying the third heart sound, which may be the most intense shock in the whole cardiac cycle. Apparently this is distinctive of adherent pericardium.

Thrills, especially presystolic in time, are occasionally felt, probably owing to tugs upon strands of adhesions, but these alone are not typical.

Auscultation.—Since pericarditis is frequently (34 per cent. of Sears's cases) accompanied by various forms of valvular disease, the presence of

all varieties of valvular murmurs, especially of mitral origin, is not surprising. A presystolic rumble, probably due to the stretching of strands of adhesions by the contraction of the auricle, is occasionally heard in cases of adherent pericardium in which aortic, mitral, and tricuspid valves are normal. Sewall also reports several cases with reduplication of the first sound, which was shown at autopsy to be due to old peripheral adhesions. Professor Thayer finds the third heart sound and the corresponding protodiastolic shock and wave very distinct in adherent pericardium. This may be due to the fact that they are more easily transmitted to the chest wall, or perhaps because the filling of the heart causes sudden stretching of the adhesions.

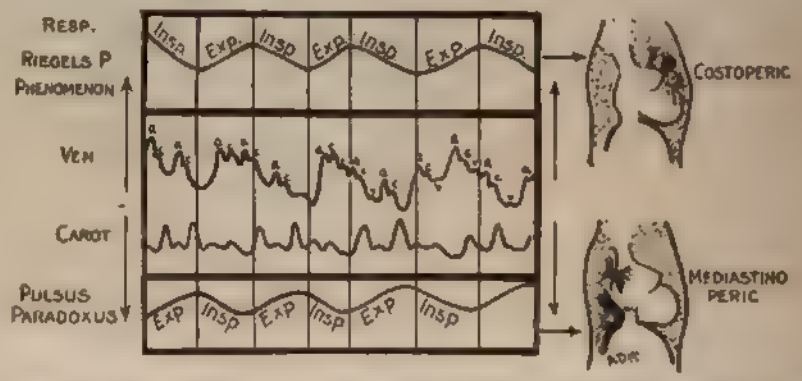

FIG. 200.—Inspiratory and expiratory dropping of beats. Riegel's pulse and the pulsus paradoxus in adherent pericardium, showing the position of the adhesions which bring the condition about. *VEN*, jugular pulse. *CAROT*, carotid pulse. *RESP*, respiration; downstrokes represent inspiration, upstrokes represent expiration. In cases with Riegel's phenomenon anterior costo-pericardial adhesions; the conditions are as shown in the diagram (upper respiratory tracing, venous pulse, carotid pulse); those with pulsus paradoxus correspond to the conditions shown by venous pulse, carotid pulse and lower respiratory tracing.

Riess' Gastric Sounds.—A very interesting sign was described by Riess in 1879, and, since it has been verified by so excellent an observer as François-Franck, merits attention. On listening over the stomach in some cases of adherent pericardium, observers have been able to hear the heart sounds loud and metallic in quality. These sounds are not much influenced by changes of position, by respiration, nor by inflation or filling of the stomach. They are probably due to adhesions to the diaphragm only, and hence, as originally stated by Riess, are not present in all cases of adherent pericardium.

Variations in the Pulse.—The pulse in adherent pericardium is usually small and rapid, generally regular, but often showing an extrasystolic irregularity which is probably due to tugs upon the strands of adhesions.

The striking and characteristic feature is the marked diminution of the pulse-wave during inspiration, amounting sometimes to the dropping of a beat during that phase. This was first noticed by Griesinger in 1854 in a case in which autopsy showed strands of adhesions about the arch of the aorta, causing kinks and stenosis in its lumen when pulled upon by the descent of the diaphragm. The venæ cavæ were also caught in dense

adhesions which strangulated them in similar manner during inspiration. Both inflow and outflow of blood were therefore hindered in that phase, hence the diminution of the pulse. The same condition was studied by Hoppe and later by Kussmaul (1873), since whose report it is known as the pulsus paradoxus. It is not entirely pathognomonic of adherent pericardium or even of pericarditis in general, occurring with open ductus arteriosus Botalli (François-Franck, see page 450) and in many normal or neurasthenic individuals (Reichmann), though in these the diminution does not amount to complete dropping of beats during inspiration.

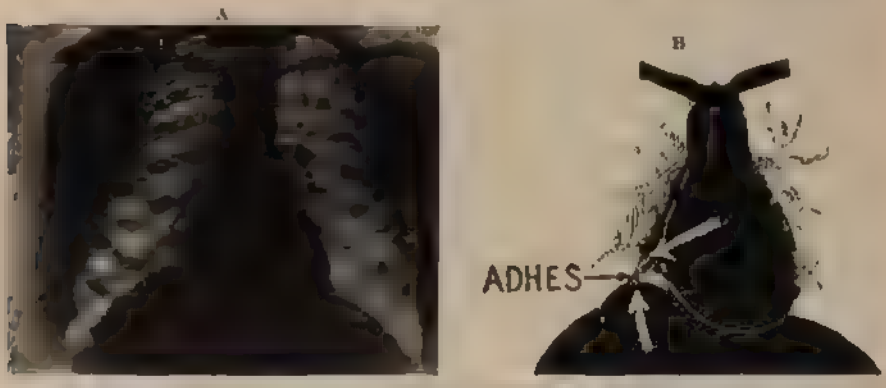

FIG. 291.—A, Radiograph of a case of adherent pericardium. (Kindness of Prof. C. M. Cooper.) B, Diagram illustrating the condition seen in A, showing the pericardium pulled outward to the right and a portion of the diaphragm pulled upward by the adhesions (*ADHES*).

The occurrence of exactly the opposite condition of the pulse, namely diminution of the wave and impulse during expiration, has been described by Riegel in cases in which autopsy showed pleuropericardial adhesions upon the anterior surface of the heart. Riegel believes that the relaxation of the lung during expiration pulls the heart upward and produces a fall of pressure from displacement of the latter.

Rosenbach has been able to show experimentally that when the heart was displaced by an inflated rubber bulb the vena cava became kinked and the pulse became smaller and blood-pressure fell. When this displacement occurs during inspiration from downward traction, a pulsus paradoxus results; when it occurs during expiration, Riegel's phenomenon occurs.

Apart from these respiratory variations the blood-pressure shows no special features, being usually low in uncomplicated cases; but it is frequently normal from compensatory vasoconstriction and increased cardiac effort, and occasionally high in the nephritic and uræmic cases.

X-ray Examination.—The demonstration of pericardial adhesions by means of the Röntgen rays was first made by Moritz Benedikt in 1897. Some question was thrown upon his methods by the criticism of F. Moritz (1900), showing that normal shadows along the edge of the cardiac and liver shadows may simulate adhesions. These objections were obviated by Stuertz, who reported five cases in which the presence of adhesions was demonstrated not only by suspicious shadows through the lungs and along the edge of the pericardium, but also by the fact that the margin of the pericardium at these points was pulled downward or outward when the structures were rendered tense in inspiration. Some areas were also shown

to be quite fixed during respiration. Stuertz's observations have been confirmed by Lehmann and Schmoll and by Dr. C. M. Cooper, to whom the writer is indebted for the X-ray shown in Fig. 291.

The special value of the X-ray examination lies in the fact that it reveals the mediastinal and diaphragmatic adhesions with accuracy, and, by demonstrating the points at which the fixation and tension are greatest, points out the path for operative interference.

Abdomen. The abdomen is often negative, but enlargement of the liver and spleen and ascites are frequent, as has been shown by Weiss in 1876.

Case of Adherent Pericardium.

The following very typical case was under the writer's care in the City and County Hospital of San Francisco. (As the original history was lost, these notes are taken from the article of Lehmann and Schmoll, who have previously published the case.)

L. A., engineer, 23 years old, entered the hospital complaining of headache, nausea, and shortness of breath. He had had rheumatism six years before admission and then had pain over the heart. Since then he had had two attacks. During the past few years he has been subject to periods of heart failure with dyspnœa, during which he is frequently depressed and sometimes even maniacal.

The patient's lips, ears, and extremities are deeply cyanotic. The pulse is irregular with numerous extrasystoles, many of them ineffectual. The apex impulse (systolic protrusion) is visible in the sixth interspace 3 cm. outside the mammillary line, beyond which there is a well-marked systolic retraction of the interspaces in front and back. There is also systolic retraction of the ribs and costal margin (Broadbent's sign). The apex is fixed and does not move with change of position, but the area of flatness changes during respiration (movement of the lung border). There is well-marked pulsation over the right ventricle. Dulness extends above to the third rib and 3 cm. to the right of the right parasternal line. A loud presystolic rumble and a loud systolic murmur are heard over the apex. The second pulmonic is markedly accentuated. Both sounds are heard with the extrasystoles.

The lungs are clear except for dulness and bronchovesicular breathing at the left base behind.

The liver is greatly enlarged and readily palpable, but there is no pulsation. There is some œdema of the feet.

Clinical diagnosis: Left-sided pleurisy, adhesion of the pericardium with the posterior surface of the heart, mediastinum, and diaphragm, mitral stenosis and insufficiency.

Examination with the fluoroscope showed the heart to be dilated to right and left. There was a marked angular protrusion along the right border of the cardiac shadow. In this region the outlines of the shadow are less sharply defined than usual, merging into the liver and vertebral shadows. The diaphragm is equally high on left and right, moving less on the latter.

The patient's condition did not improve under rest and digitalis. He often had intense precordial pains. On one occasion he was subject to definite hallucinations, imagining that he saw lions, tigers, and other brightly colored wild animals springing to and fro upon the floor of the ward and over his bed, though he was at the time otherwise rational, and even realized that it was an hallucination. He was placed in a solitary cell for twenty-four hours at his own request, for fear of doing personal violence to the persons about him.

His condition became so much worse that cardiolysis was decided upon as a last resort and was performed by Professor Stillman. The ribs were resected over the precordium and the pericardium opened in exploration. The heart was everywhere covered with adhesions, which over the anterior surface of the heart consisted of strands about an inch long. There was no fibrinous exudate and no fluid. The patient took the ether badly and became extremely cyanotic. The shock of the operation did him evident harm, for during his entire sojourn after that he felt even worse than before. The wound itself caused him no trouble and healed per primum. The patient left the hospital three weeks after the operation, in spite of advice.

Pericarditic Pseudocirrhosis of the Liver (Pick), and Polyserositis from Adherent Pericardium (Cabot).—Hutinal in 1895 described a form of liver cirrhosis of cardiac origin (cirrhose cardiaque). Friedel Pick (1896) in Pribram's clinic called attention to a very interesting clinical condition which is not infrequently encountered, but whose nature is often overlooked. This is seen in certain cases which run the course of a primary hepatic cirrhosis, beginning with ascites, enlargement of the liver, slight jaundice, general weakness and dyspnœa, but devoid of any special cardiac features. Occasionally there were also enlargement of the superficial veins of the abdomen and œdema of the feet. The first and second cases were considered clinically to be primary cirrhosis of the liver, and the discovery of adherent pericardium at autopsy came as a surprise. In his third case adherent pericardium was carefully sought for and found, and the diagnosis was correctly made. Death occurred in two to four years after onset of symptoms. The pericardia in these cases were found to be completely or almost completely adherent, the rest of the heart normal. The livers showed both interlobular cirrhosis and chronic perihepatitis (nutmeg liver, Curschmann), the peritoneum was thickened, and chronic perisplenitis was present. An example of this condition is found in the case of J. M. C. cited on page 272, in whom the presence of adherent pericardium was not suspected during life.

Fig. 202.—Case of pericardial pseudocirrhosis. After Cabot: *Bost. M. and S. J.*, 1898, cxxxviii.

In 1898 R. C. Cabot described a similar case. Flesch and Schossberger find the condition not infrequent in children, presenting the superficial manifestations of a primary cirrhosis without the presence of alcohol and syphilis as etiological factors. On careful examination the presence of adherent pericardium is readily detected by its usual signs.

Flesch and Schossberger were able to reproduce the condition experimentally in dogs. They produced pericarditis by injections of tincture of iodine into the pericardial cavity and allowed the animals to recover during which period adherent pericardium occurred. After a few months ascites and œdema set in and the animals died. Their results have been confirmed by O. Hesse, who has also produced cyanosis and cirrhosis of the liver by suturing the inferior vena cava to the diaphragm.

Another point in the differential diagnosis from true primary cirrhosis is the fact that the veins of the arms and neck are usually enlarged to almost the same extent as those of the portal system, showing that the stasis is not confined to the latter. There is no caput medusæ.

TREATMENT.

The treatment of adherent pericardium may be both palliative and operative. The palliative treatment is simply the general treatment for cardiac weakness: rest, diet, and cardiac stimulants, strychnine and digitalis, during the onset and acute stages; careful graduated exercises and training during the period of relative freedom from symptoms.

It is impossible to remove the condition, and the therapy must be simply so directed that that which cannot be cured may best be endured.

Anæmia should be treated with iron, exposure to infection avoided, and general hygienic conditions maintained. For reasons mentioned above, these precautions should be carried out even more carefully than for simple valvular disease.

Surgical Treatment (Cardiolysis). — In 1902 Brauer, of Heidelberg, introduced a simple method of treatment which promises to revolutionize the therapy of adherent pericardium. Brauer proposed "to relieve the heart functionally by breaking the strong bony ring of ribs, not by a severe operation with the breaking up of extensive adhesions but only by substituting a soft covering for the natural bony covering of the heart. . . . . On account of the tremendous strain upon the heart, due to traction on the chest wall, we foresaw a danger in operation under narcosis. . . . . The operation was tried upon a patient with adherent pericardium, broken compensation, ascites, and œdema. Segments of the third, fourth, and fifth ribs 7 to 9 cm. in length were resected under light narcosis, the periosteum being carefully removed. The patient made an uninterrupted recovery. His pulse soon became stronger and more regular, the ascites and œdema disappeared, and he was able to do heavy work without symptoms. The pulse still remained irregular"

Brauer reported two other cases with equally good results, and these have been confirmed by Beck, Umber, Meyer, Westfeld, Wenckebach, and others. Brauer particularly states that he does not attempt to break up the adhesions, as Delorme and Carl Beck have proposed, since he believes that this operation is too severe and that the adhesions would form again too rapidly, although he states that, in individual cases, this might be done besides his operation.

As regards the indications for cardiolysis, it would appear that, since the adherent pericardium cannot otherwise be relieved, this operation is worthy of trial whenever symptoms of cardiac weakness occur and recur in a patient with well-marked adhesions to the chest wall (tugging in of the lower ribs, fixation of the left border of flatness on inspiration, immobility of the apex) and recur in spite of general cardiac hygiene. It is not necessary to wait for the complete cardiac break-down to prophesy that this must sooner or later occur in such a case, and to see that the sooner the work of the heart is relieved the longer will be the life of the patient. Moreover, it is evident that if the operation is performed between attacks of cardiac overstrain, the patient is in better condition to withstand the shock of the operation and the danger of the latter is diminished. If the cardiolysis is not performed until the patient's heart has almost completely given way, as in the case of the patient with the visual hallucinations referred to above, he can scarcely fail to suffer from the shock of the operation, but even in such cases Brauer's results have been striking, and, since there is no other mode of relief, operation is warranted.

It must be confessed that in such cases the manner in which the anæsthesia is administered determines a large part of the shock from the operation, and may prove a decisive factor in the outcome. The selection of the anæsthetist constitutes no small part in the management of the case

The question also arises whether operation should be advised in children or adolescents whose pericardia are adherent to the chest wall, but in whom, owing to the flexibility of the latter, the symptoms do not as yet demand operative interference. In this regard each case must of course be decided upon its own merits, but it is evident that as age advances the rigidity of the ribs is bound to increase and the strain upon the heart proportionately. If the case remains relatively free from symptoms as age advances, it should be left alone; but if the progress of the second or third decade brings with it increasing cardiac symptoms or the signs of pericarditic pseudocirrhosis, the question of early cardiolysis should be seriously considered. Since there is no hope that children will "outgrow" an adherent pericardium, it should be relieved as much as possible before the strain has ruined the heart muscle. When valvular lesions are present, especially mitral stenosis, the danger from operation is of course greater, but in the hands of a skilful surgeon this is much less than might be expected and is probably less than that in pericardiotomy for purulent pericarditis.

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XIII.

WOUNDS OF THE HEART AND CARDIAC TRAUMA.

Hippocrates and Celsus, Paul of Ægina, Roland, Lanfranc, and other writers of antiquity taught that wounds of the heart were followed immediately by death; but Ambroise Paré (1552) saw a gentleman of Turin "who, although wounded in the heart during a duel, was able to pursue his antagonist 700 feet before he dropped to the ground and died." Muler (1641) treated a soldier who lived for fifteen days after sustaining a wound of the heart,—an observation so unheard of at the time that he had the autopsy protocols signed by the commander of the garrison! Aprilis (1680) describes a wound of the right auricle, after receiving which the man had lived for five days.

The results of modern times were summed up by G. Fischer in 1867 (351 cases) and Loison (1899) (277 cases). Fischer found the wounds occurring with the following frequency:

	Death within a few minutes.	Recovery.
Right ventricle	107—21.9 per cent.	6
Left ventricle	95—25. per cent.	6
Both ventricles	24—34. per cent.	2
Right auricle	28—25. per cent.	
Left auricle	13—38. per cent.	
Apex	12	4
Base	1	1
Septum ventriculorum	6	1
Whole heart	15—62. per cent.	1
Left heart	5	
Right heart	3	11
Coronary artery	1	1
Pulmonary artery	1	
Not specified	40	17
	351	50 (11.2%)

In 452 cases there were 50 (12 per cent.) of spontaneous recovery.

EXPERIMENTAL SURGERY.

Elsberg in 1899 made a very careful study of wounds experimentally produced in the rabbit's heart. He found that those produced during systole, when the heart fibres are shortened, become enlarged during diastole and hence bleed more than wounds of corresponding size produced during the latter phase. Wounds that completely penetrate the heart wall bleed more than those which do so partially. Those which enter perpendicularly bleed more than those which penetrate obliquely, for in the latter case the walls form a valve-like approximation during systole. Indeed Prof. Barker and the writer have produced oblique wounds penetrating the entire wall of the dog's ventricle, which scarcely bled at all.

SYMPTOMS.

The symptoms accompanying a wound in the thorax which suggest a wound of the heart (intrapericardial pressure) are those of angina pectoris—pain down the left arm, a feeling of precordial oppression and precordial pain, especially marked on expiration. Pressure upon the precordium increases these pains. There is shortness of breath. Occasionally there are abdominal pain and spasm of the abdominal muscles (Rehn). As Fischer pointed out, pain is also felt about the external wound, but as a rule not in the heart itself. Even probing of the heart wound, while it may give rise to weakness and syncope, is not accompanied by pain. Thus, one patient whose left ventricle had been wounded thought that the knife had only gone through his clothes. Blood is often found spurting from the wound with a well-defined pulsation. Sometimes it is foamy and mixed with air, indicating that the lung has been penetrated.

PHYSICAL SIGNS.

The area of cardiac dulness is increased or is replaced by tympany (pneumo-hæmopericardium). The heart sounds are replaced by loud churning or water-wheel murmurs. The blowing murmur caused by the jet of blood passing out of the heart may also be distinguished.

The pulse becomes small, weak, rapid, and finally imperceptible.

Whenever time warrants, an X-ray examination should be done at once, and the bullet or foreign body located. This may sometimes be very exactly done by means of stereoscopic pictures and greatly simplifies the operation.

TREATMENT.

Operative interference in the treatment of wounds of the heart was first proposed by Rose, who confined himself to opening the pericardium and removing the blood that compressed the auricles. This procedure was often of benefit and even effected cure in cases where bleeding ceased spontaneously, but when the heart continued to bleed it was of no avail.

Up to this time it had been thought, in spite of the experiments of physiologists, that suture of the heart wall itself would be accompanied by instant death. But in 1895 Salomoni and Del Vecchio demonstrated that wounds in the heart of the dog could be successfully treated in this manner, and in 1896 Cappelen, Farina, and Rehn sutured the heart wall in man. The passing of the sutures had no ill effects. Cappelen's and Farina's patients died a few days later from secondary causes, but Rehn's patient, who had received a stab wound in the right ventricle, operated on forty-eight hours after the injury, recovered, and thus a revolution in cardiac surgery was made. Rehn had demonstrated that wounds of the heart could and should be successfully explored and sutured like wounds of other viscera.

If the patient is in severe collapse from loss of blood, an intravenous infusion of warm salt solution (37° C.) should be begun at once while the operators are hastily cleaning and disinfecting the field of operation. As a last resort a direct arteriovenous transfusion into the veins of the arm

may be made from another individual by the method of Crile, Buerger, or Hartwell while the operation on the heart is going on, and some exsanguinated patients may thus be saved.

Operative Procedure. — The incision should be sufficiently large to admit of a satisfactory exposure. A flap is made in the chest wall over the point of penetration, usually including two ribs and three interspaces. The flap adopted by most operators is horizontal U shaped with bifurcations pointing to either left or right, the connecting bar passing through either sternochondral or costochondral articulations. Occasionally the

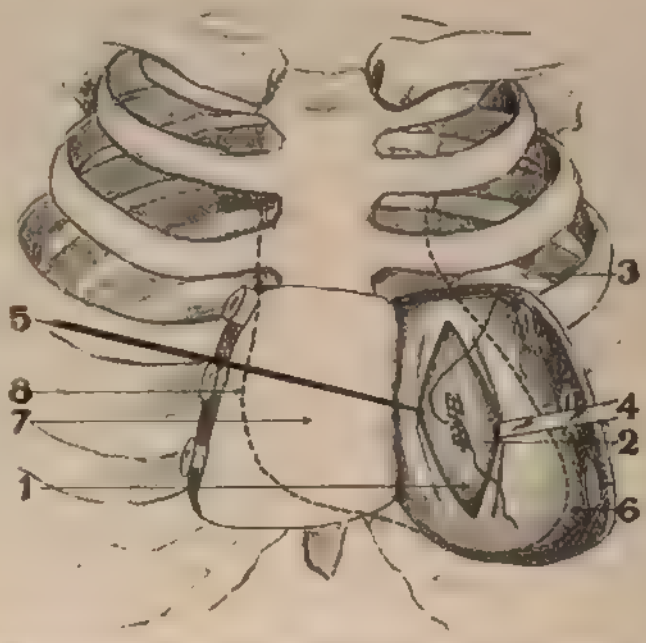

FIG. 294. — Exposure of the heart for suturing a wound. (After G. T. Vaughan, J. Am. M. Assoc., 1909, liii, 1, heart, 2, deep sutures, 3, superficial sutures, 4 and 5, retractors on the pericardium, 6, left pleural line; 7, flap of chest wall including the fourth, fifth, and sixth ribs, 8, heart, outlined by broken outline.

form is that of an upright or an inverted U, a ⊢, or an H. If the wound is near the sternum and has not already penetrated the pleura, that cavity should not be opened, and the ⊓ or ⊢ shaped flap is the best; but if the wound has pierced the pleura, any convenient exposure may be adopted. The incision through the pectoralis major should be parallel to its fibres which may be retracted. The sternochondral or costochondral articulations are cut through, the ends of the incision prolonged along parallel to the ribs, and the flap forcibly reflected back, fracturing the costal cartilages to permit a wide opening. If the pleura has not been penetrated, it should be pulled toward the outer edge of the wound with retractors. A free incision should be made into the pericardium, the pericardial cavity emptied of clots, the wound in the heart located, and sutured with a fine curved needle and silk thread. In passing the sutures the heart wall may

be grasped with forceps without danger, the irregularity which accompanies the passing of the needle representing merely a few extrasystoles resulting from the irritation, and passing off rapidly. Elsberg never observed sudden stoppage of the heart and fibrillation following the insertion of sutures. The writer, after several hundred experiments upon exposed dogs' hearts, is able to confirm these statements of Elsberg. Elsberg states that the interrupted suture is preferable to the continuous, for, though it takes longer to apply, it injures fewer muscle fibres and is more certain to hold. The sutures should be tied *during diastole*; these do not tear out as readily as sutures tied during systole. In tightening the sutures the two serous surfaces of the wound should be pushed in so as to be brought into apposition. The surfaces unite by the usual growth of fibrous tissue. The nuclei of the muscle cells near the wound seem to be increased in number, and there is some amitotic and mitotic division but no definite regeneration of muscle.

Control of Hemorrhage. - When the bleeding was so profuse that death seemed imminent, Elsberg found it necessary to adopt provisional means for stopping bleeding while putting in the sutures. For this he used a hastily placed tobacco-pouch suture, or even a ligature about the whole heart just above the wound. (This does not apply, of course, to wounds in the upper half of the ventricles.) He was then able to place the sutures bloodlessly, after which the provisional ligature was removed. In this way he was able to suture tremendous wounds (2 cm. in a rabbit's heart, corresponding to about 10 cm. in the human heart), with 66 per cent. of recoveries.

Large wounds of the auricle may be more difficult to control. Sauerbruch recommends stopping the bleeding by gently compressing the auricle between the middle and ring fingers while grasping the point to be sutured between the index finger and thumb. Rehn finds that with some care a ligature may be placed about the auricle to still the bleeding while the sutures are rapidly put in, but there is danger of death from fibrillation if the circulation is completely cut off. The writer has been able to control the hemorrhage from quite large wounds in the dog's heart for over ten minutes by holding his finger gently against the wound. The heart's action was not weakened by this procedure, nor did it become irregular; and sufficient time was gained to lay the sutures carefully. This was found to be more bloodless, and for large wounds more convenient, than Elsberg's method of laying temporary sutures.

If possible the bullet should be removed unless it is too deeply imbedded in the cavity of the heart. Under these circumstances it may be left at least for a subsequent operation, as it often becomes encapsulated and may do no further harm. All operators agree that operation in the Sauerbruch negative pressure chamber or with Brauer's positive pressure lessens the danger of pneumothorax, and is therefore advisable when it requires no delay. It is particularly useful when the wound is about to be closed, to prevent the continuance of the pneumothorax.

As regards the question of drainage, each individual case must be decided on its own merits. It is, of course, important to prevent sepsis, purulent pericarditis, and pyopneumothorax. When the pleura has not

been pierced, the pericardium may be closed in a large number of cases without drainage (Rehn's statistics show 4 cases—3 cures, 1 death—without drainage of pericardium; with drainage, 5 cases—5 deaths; perhaps, however, drainage was used in only the more severe cases). When the pleura has been pierced, it should usually be drained. Whenever bits of cloth, dirt, etc., have entered the wound, it should always be drained.

Before closing the wound the pericardial cavity should again be explored to see that no other wounds in the heart wall or vessels have been overlooked.

Occasionally large branches of the coronary arteries are found to be pierced and must be ligatured. This is necessary, and, as shown by Porter and Baumgarten (see page 280), is not always fatal, as there is a certain amount of collateral circulation, but sudden death may result during subsequent excitement, so that in such cases more prolonged rest is advisable than in cases of simple suture. It is worthy of note, however, that this complication is not mentioned in the twelve cases of late results compiled by Rehn.

After closure of the wound, with or without drainage, administration of urotropin is probably advisable, since Crowe has found that it is excreted in the pleural and pericardial fluids in a concentration sufficient to inhibit the growth of bacteria, and Bernheim believes that its use increases the resistance of these membranes (in dogs at least) to infection. It has, moreover, no harmful effects.

Results of Operation.—Since Rehn's first operation a large number of cases have been reported. In 1907 he was able to collect statistics of 124 cases—49 recoveries (39.5 per cent.), 75 deaths (60.5 per cent.). In this series there were only 15 cases of gunshot wound, but in a series of 30 cases of the latter compiled from the series of Ricketts, Borchardt, and Rehn, there were 14 recoveries (46.6 per cent.) and 16 deaths (53.4 per cent.). Of the 75 deaths in Rehn's series 16 died on the operating table, 17 died of loss of blood and collapse within two days, 30 died of infection (purulent pericarditis and empyema). In many cases the haste of operation prevented disinfection of the field. One patient (Gerzen's) died of sudden hemorrhage on the fifty-third day. Rehn also collected reports of 12 cases from nine months to ten and one-half years after operation. In nine examination of the heart was negative; in three there was slight dilatation. There were costopericardial adhesions in 5; 9 were absolutely free from symptoms, 2 had pains down left arm; 1 precordial pain. Only one had symptoms of definite cardiac weakness.

G. T. Vaughan has recently summarized and tabulated 150 cases operated on between 1896 and 1909, of which 51 (34 per cent.) recovered, a striking contrast to the 12 per cent. of recoveries in the earlier years from which Fischer's series was taken.

NON-PERFORATING INJURIES.

Injuries of the chest wall which do not enter the pericardium, such as blows upon the chest, frequently produce secondary lesions of the heart and pericardium, which have been mentioned in previous chapters.

The first case of cardiac disease from contusion was recorded by Blancard in 1688 and is very typical. The patient was a peasant 45 years of age, previously healthy, who was run over by a hay-cart. He did not sustain any fracture, but suffered from pain in the chest, dyspnœa, then fever, delirium, and died 11 days later of purulent pericarditis and myocarditis. Similar cases were recorded by Bonetus (1700), Akonside (1766), and numerous other writers both ancient and modern.

Bernstein in 1896 was able to collect 126 cases from the literature. In autopsies upon 42 of these cases there was found

Endocarditis alone	16 times
Myocarditis "	0 "
Pericarditis "	10 "
Endo- and myocarditis	4 "
Peri- and myocarditis	5 "
Endo- and pericarditis	5 "

The signs and symptoms appeared:

Immediately after the trauma	67.6 per cent.
Within one month	17.5 " "
Within one year	4.7 " "
Later than one year	7.1 " "
Time not given	3.1 " "

G. Fischer gives a list of the causes of traumatic rupture of the heart in his series:

Run over by or crushed between wheels of wagon	21
Crushed by machinery	4
Falls from considerable heights	13
Falls from heights of 10 feet or less	7
Struck by falling objects	6
Kicked in chest	4
Hurled against wall	2

Külbs has recently investigated the subject experimentally. The results in 23 animals within 12 days of the injury were:

Hemorrhages into the heart valves (1 rupture of an aortic valve)	17 times
Subendocardial or subpericardial hemorrhages	10 times
extensive hemorrhage into the septum	3 times
Pericardial hemorrhages	10 times
Hemorrhages from lungs	6 times

There was polymorphonuclear infiltration and disintegration of muscle fibres in the vicinity of the hemorrhages.

The symptoms and signs of these conditions following trauma do not differ from those in similar lesions due to other causes, and have been considered under those heads.

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XIV.

ANEURISM.

Aneurism (aneurysm) (Greek ἀνεύρυσμα a widening out) = a dilatation of artery or veins (Galen)

An aneurism is a blood-containing tumor whose walls are formed by the walls of a blood-vessel and whose cavity is in direct connection with the blood-vessel from which it arises (Osler).

Historical.—Hippocrates (430 B. C.) and the early Greek writers do not seem to have been familiar with aneurism, but its occurrence and nature were well known to Rufus and Galen (A.D. 131–201), who recognized two forms "one from dilatation, the other from wounding of a vessel," usually from venesection followed by sepsis.

FIG. 295.—Specimen of a large aneurism. (After Hough.)

Vesalius (1543) was the first to recognize aneurisms within the thorax and abdomen and was even able to make the diagnosis of thoracic aneurism during life. Ambroise Paré (sixteenth century) recognized the existence of "aneurism by anastomosis, rupture, erosion, and wound along with the frequency of thrombosis within the sack." He was also the first to suggest that venereal disease was a factor in the genesis of aneurism. The rôle of syphilis was demonstrated definitely by Lancisi (1728). The next great step was made by Scarpa (1805), who demonstrated that the most important mechanical factor was weakening of the middle layer of the arterial wall, a fact which has furnished a basis for the more modern pathology of aneurism.

CLASSIFICATION OF ANEURISM.

It is extremely difficult to make a satisfactory classification of aneurisms, but the following, which is based upon that of Osler, may suffice for most purposes:

1 True aneurism (aneurysma verum, aneurysma spontaneum), in which one or more of the coats of the artery form the walls of the tumor.

A. Dilatation aneurism

(a) Sacculated, in which the bulging or out-pocketing of the walls does not embrace the whole circumference of the artery and is sharply localized

(b) Fusiform (or cylindroid) aneurism, in which the dilatation occurs over a larger area of artery whose entire circumference is involved in the dilatation.

2. Dissecting aneurism, in which the coats of the artery are separated and a new cavity (sometimes lined with endothelium) is formed between these layers (usually between media and adventitia).

3. False aneurism, following wound or rupture of an artery, consisting of a perarterial hæmatoma, all the coats of the artery having been penetrated.

4 Cirsoid aneurism or telangioma, a tumor consisting of a large number of tortuous arteries which are continuous with the artery from which they arise.

5 Arteriovenous aneurism, a communication between artery and vein, either direct, aneurismal varix, or with the intervention of a sac, varicose aneurism.

ARTERIES AFFECTED.

By far the most common forms are the true aneurisms, fusiform and sacculated. The relative frequency in the various arteries is shown in the following statistics of 530 cases (Crisp):

Thoracic aorta	175
Popliteal artery	137
Femoral artery	66
Abdominal aorta	59
Carotid artery	25
Subclavian artery	23
Axillary artery	18
External iliac artery	9
Cerebral artery	7
Common iliac artery	2
Posterior tibial artery	2
Gluteal artery	2
Pulmonary artery	2
Brachial artery	1
Subscapular artery	1
Ophthalmic artery	1

OCCURRENCE.

According to a large set of statistics compiled by Richter and by Arnsperger, aneurism of the aorta represents one of the not infrequent causes of death, 0.6 per cent of total mortality (Emmerich), Brodier 1.2 per cent., Muller 1.49 per cent.; in American cities 0.6 per cent., Philadelphia 0.6 per cent., St. Louis 0.2 per cent.

According to Gibbons and Richter the percentage of deaths from aneurism in San Francisco from 1866 to 1870 (1.35 per cent.) was much greater than elsewhere in the United States. Dr. Gibbons has informed the writer that aneurism at that time was particularly common among stevedores, who formed a considerable percentage of the population and in whom syphilis, alcohol, and hard work were ever present factors. With the passing of the adventurer and the stevedore as important elements in the population, the percentage of aneurism in San Francisco has diminished, being 0.90 per cent. in 1880–1884, 0.12 per cent. in 1890–1894, 0.33 per cent. in 1900–1901 (Gibbons).

On the other hand, in communities where syphilis is common the frequency of aneurism increases. Thus, it is eleven times more common in the British Army in India than in the civilians at home, and much more common

in the British than in the Austrian and German armies, where venereal disease is five times less prevalent.

Aortic aneurism is much more common in men than in women.

Crisp	67 women out of	551 cases
Agnew	26 women out of	269 cases
Lisfranc	13 women out of	154 cases
Richter	58 women out of	736 cases
	164 women out of	1810 cases

9.05 per cent., or 1 in 11.

On the other hand, 48 per cent. of cases of carotid aneurism and 66 per cent. of dissecting aneurisms occurred in women.

As regards age Crisp's cases were distributed as follows:

1 to 10	1	50 to 60	65
10 to 20	71	60 to 70	25
20 to 30	51	70 to 80	8
30 to 40	198	80 to 90	2
40 to 50	129	90 to 101	1

59 per cent. between the ages of thirty and fifty.

As regards site Lawson gives the following figures:

Ascending aorta	34. per cent.
Arch of aorta	34.8 per cent.
Descending aorta	17.4 per cent.
Abdominal aorta	13.8 per cent.

Hare and Holder find in 953 cases collected indiscriminately:

Ascending	570 = 60 per cent.
Transverse arch	104 = 10.6 per cent.
Descending	110 = 11.5 per cent.
Unclassified	169 = 17 5 per cent.

figures which are certainly unusually high for the ascending portion.

Aneurisms are by no means always single, but may sometimes be multiple. Two, three, "or even a score" may appear along the course of the aorta, or numerous aneurisms may be present in the peripheral arteries. The condition is simply the manifestation of the generalized action of the factors of sclerosis and blood-pressure on the arterial walls, and multiple aneurism formation is one of the features of experimental adrenalin aortitis (Erb, Jr.).

The symptoms, signs, and diagnosis present no specific features, except the ease with which the other aneurisms may be overlooked after one is diagnosed. Careful examination and especially fluoroscopic examination will prevent this error.

PATHOLOGICAL ANATOMY AND PATHOGENESIS.

No change of pressure that can occur during life is sufficient to dilate an artery to the proportions of even the smallest aneurism. According to the elasticity curve of Roy, the dilatation occurring between the blood-pressure of 120 and 170 mm. Hg is about 20 per cent. of the diameter of the artery, and the results of Grehant and Quinquaud show that very little further dilatation occurs if pressure is raised until the artery ruptures (at a pressure of 1680 to 4630 mm. Hg; 10 to 20 times the blood-pressure during life).

FIG. 296.—Aneurism arising just above a sinus of Valsalva. (From a specimen in the Army Medical Museum, Washington D. C. *OR*, orifice through which the aneurism is connected with the aorta, *AN*, aneurismal sac.

FIG. 297.—Aneurism of the ascending arch and innominate artery. (From a specimen in the Army Medical Museum.)

FIG. 298.—Aneurism of the transverse portion of the aorta arch penetrating through the sternum (from a specimen in the Army Medical Museum). *INNOM.*, innominate artery, *L. CAR.*, left carotid artery, *L. SUB.*, left subclavian artery, *D. AOR.*, descending aorta.

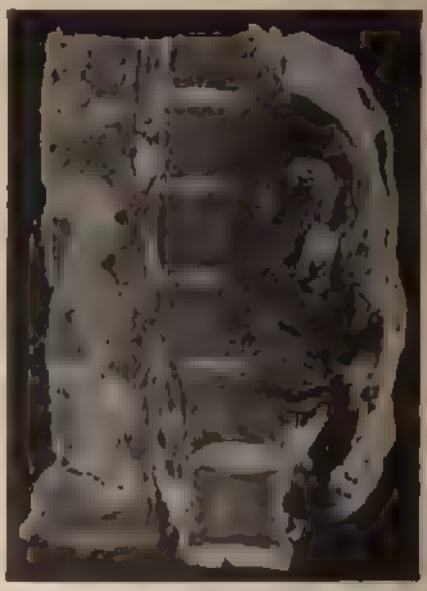

FIG. 299.—Aneurism of the descending aorta eroding the vertebræ. The sac contains laminated clot

Changes in Arterial Wall in Aneurism.—On the other hand, as was first shown by Scarpa (1805), aneurismal dilatation is always preceded by changes in the arterial coats and especially by weakening of the media. In 1875 Koester showed that this was due to localized degeneration of the elastic fibres as the result of certain inflammatory changes in the vasa vasorum of the media. "The inflammatory process begins in the vasa vasorum on the exterior of the blood-vessel, follows them perpendicularly into the muscularis (media), and distributes itself within this layer, being most intense at the places where the vasa vasorum break up into capillaries. As a result of this chronic multilocular mesarteritis, the media (muscle fibres and elastic fibres) degenerate. The intima (which may be thickened) and the adventitia unite to form a thick and very vascular membrane which forms the wall of the aneurism." Since these studies of Koester, writers are practically agreed that the degeneration of elastic tissue resulting from mesarteritis is the underlying cause of aneurism formation.

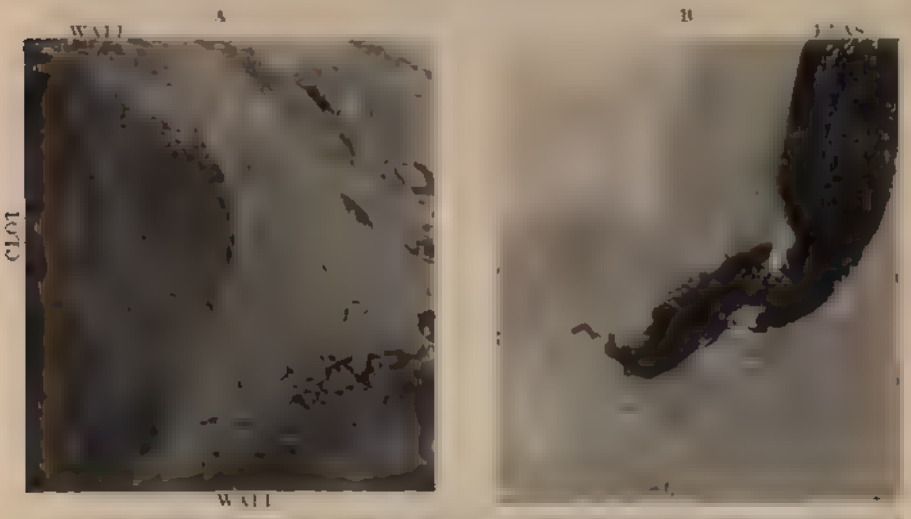

FIG. 300.—Sections through the wall of an aneurism. (Photomicrographs made by Dr. C. S. Bond.) A, Section through the wall of an aneurism showing the clot unorganized. B, Orcein stain showing the destruction of elastic tissue in the aneurism wall. Elastic tissue (*ELAS*) stained dark.

Simple arteriosclerosis in which thickening of the intima is the essential feature does not weaken the wall of the artery and plays no rôle unless the media be destroyed. This fact is further borne out by the experimental changes in the artery produced by injection of adrenalin (see page 257). The resulting lesion is a mesarteritis without changes in the intima, quite dissimilar to the ordinary arteriosclerosis of man; but aneurisms, and even multiple aneurisms, are present in a large percentage of the animals. Other toxic substances, bacterial toxins, lead, alcohol, nicotin, lactic and other acids, etc., produce these changes.

Fabris has also produced aneurisms by external cauterization of the arterial wall with silver nitrate. A local inflammation was thus set up in the adventitia and media, which resulted in degeneration of the fibres of the latter and their replacement with young fibrous tissue devoid of elastic

fibres. In a few cases there was slight intimal thickening. The resistance of such a fibrous tube is less than that of an elastic tube, and aneurismal dilatation, sometimes localized, sometimes fusiform, took place in from 20 to 25 days.

Etiological Factors.—In man the etiological factors of aneurism are those that produce mesarteritis. Chief among these is syphilis, which, as first noted by Ambroise Paré and Lancisi, is concerned in a very large percentage of the cases (Klemperer 25 per cent., Fraenkel 36 per cent., Puppe 36 per cent., Trier 40.5 per cent., Heiberg 41.87 per cent., Bramwell 50 per cent., Thieberge 50 per cent., v. Noorden 54 per cent., Gerhardt 56 per cent., Schutz 64.7 per cent., Welch 66 per cent., Etienne 69 per cent., Malmsen 80 per cent., Hanpeln 82 per cent., Backhaus 85 per cent., Heller 85 per cent., Rasch 92 per cent.). This is especially true of aneurisms occurring in young men and women, when the other factors of arteritis play a relatively less marked rôle than in later life; so that, as stated by Professor Osler, the presence of an aneurism in a man or woman under thirty is almost to be regarded as presumptive evidence of syphilis. Moreover syphilitic aortitis is often most intense in the first part of the ascending aorta, hence the commonness of the lesion at this site (Heller). It must be added, however, that, though the careful researches of Ophuls have failed to substantiate this general belief, a positive Wassermann reaction is usually obtained in such cases.

Other factors are alcohol, hard work, lead poisoning, tobacco, gout, nephritis, and especially the infectious diseases. Trauma (blows, gunshot and knife wounds, etc.) furnishes a frequent cause for aneurisms of the peripheral arteries and abdominal aorta, but is much rarer in thoracic aneurisms. Cases like that described by Hirsh and Robins show, however, that it is a factor to be reckoned with. The relative importance of these factors is shown by the figures of Etienne, who found syphilis as a cause of 166 out of 230 aneurisms, while alcoholism was present in only 28. Moreover, according to Hamilton, aneurisms are extremely rare in sanitaria for alcoholics. They are, however, most important contributory causes, not only increasing the arteriosclerosis but, by raising the blood-pressure, increasing the liability to dilatation. Thus, aneurisms, according to most writers, are particularly common in syphilitics who perform hard work. This is exquisitely shown in the colored patients at the Johns Hopkins Hospital, in whom syphilis is very common and who, as a rule, perform hard work. Among these persons aneurisms are between five and ten times as common as in the patients in the white wards of the same hospital. The sudden rise of blood-pressure which occurs during lifting and heavy strains (cf. page 132) is a particularly important predisposing factor, and the patient often notices that his first symptoms occurred at the time of a heavy muscular strain or began just afterwards.

EMBOLIC AND MYCOTIC ANEURISMS

A somewhat rarer form of aneurism, described by Tufnell (1853), Ogle (1860), Church (1870), Smith (1870), Poulick (1875), and Weinberger (1907), is the so-called embolic or mycotic aneurism, which arises especially

during the course of acute septicæmias, of puerperal, arthritic, and influenzal origin. Septic emboli become lodged astride of the bifurcation of the smaller arteries, causing necrosis of the neighboring portions of the arterial wall, which may protrude or form a true aneurism, or may rupture into the surrounding tissues, forming a false aneurism. These arise acutely during the course of the febrile diseases. They are usually multiple and are confined to the smaller arteries, while the sclerotic aneurisms are more common in the larger arteries.

DEVELOPMENT OF THE ANEURISM.

Once formed, the aneurismal sac expands progressively, usually pushed outward from the artery along the lines of the least resistance until it meets with some obstruction. The higher the blood-pressure the more rapid is the dilatation. When pointing freely into the thoracic cavity, it may expand until it fills almost the entire half of the cavity before rupturing. On the other hand, if the proliferation of connective tissue in the wall of the aneurism does not keep pace with its growth, or if local necrosis from infection, pressure, or irritation takes place, a secondary bulging will take place at this point, and it finally ruptures there. A rupture is especially precipitated by high blood-pressure, such as occurs on exertion, and sudden deaths from this cause are quite common in aneurism.

When the wall of the sac presses upon neighboring tissues it begins to erode them. The pressure acts in the following way. First, it cuts off the blood supply to the neighborhood because the pressure within it (aortic pressure) is greater than that in the smaller blood-vessels. Secondly, necrosis of these tissues results from this compression. Thirdly, the products of necrosis are absorbed by the cells in the tissues of the very vascular wall of the aneurism as fast as they are produced. Bone tissue too is absorbed by the activity of the osteoclasts, and the wall of the sac thus advances gradually through the chest wall very much as a tumor might do, though without the intervention of abnormal cells. Thus, the aneurism eats its way through muscle, cartilage, bone, nerves, and skin, and also through the walls of the other vessels (pulmonary artery, vena cava, etc.), bronchi, and œsophagus, always forced onward in a straight line by the arterial pressure in the aorta. Hence, aneurisms usually point in the direction given them by the impact of the blood stream; those of the ascending aorta pointing to the right, those of the arch pointing upward, those of the descending arch pointing backward and to the left (Fig. 301). However, resistance of surrounding tissues, and especially local thinning of the aneurismal wall, may cause its course to be deflected somewhat from these typical directions.

Rupture. The excessive thinning which results in perforation frequently occurs when the sac has just penetrated the wall of one of the surrounding structures (bronchus, œsophagus, etc.), no doubt from the presence of local infections within their lumina, and sudden death may result from hemorrhage. Or, on the other hand, small hemorrhages may occur from the erosion of smaller bronchial or œsophageal arteries (see page 529) or through the wall of the aneurism without any such immediate results.

The growth of an aneurism after penetrating the chest wall is well shown by the outlines in Fig. 302. The sac becomes larger and larger, secondary sacculations appear upon its surface (Fig. 302), and over these the thinned skin becomes smooth, tense, glossy, and finally of a reddish

FIG. 301.—Composite figure showing the relations of various aneurisms to surrounding structures (Schematic). *OES*, œsophagus. *AN SUBCL*, aneurism of the subclavian artery. *SUP. V. C.*, superior vena cava. *AN INNOM*, aneurism of the innominate artery, pointing through the skin. *AN TR*, aneurism of the transverse portion of the arch. *PHREN*, phrenic nerve. *RET LAR*, recurrent laryngeal. *L. VAG*, left vagus. *D.A.*, ductus arteriosus Botalli. *AN P.A.*, aneurism of the pulmonary artery. *LBR*, left branches. *AN SIN VALS*, aneurism arising from a sinus of Valsalva. *AN R. COR* and *AN L. COR*, aneurisms of right and left coronary arteries. *AN R.A.*, aneurism of right auricle. *AN L.V.*, aneurism of left ventricle. The arrows show the directions in which the aneurisms usually point.

or brawny hue. The whole process usually requires a few months, but it may occur more rapidly. On the other hand, in a case described by Hirsh and Robins the pulsating tumor upon the chest remained practically unaltered in size for twenty-five years, during which the patient continued to do heavy work. Finally, however, a stage is reached at which a small perforation appears, oozing blood, and soon after, with a rush of blood like

the bursting of a dam, the aneurism ruptures and the patient bleeds to death within a few minutes.

The rupture into the bronchus, trachea, or oesophagus proceeds in the same way. There is usually a slight premonitory hæmoptysis. This generally occurs a few days before death, but may not occur until a few hours before the final rupture; or, as in the case reported by Clarke, it may be present for months. At the final rupture the blood spurts out of the patient's mouth and nose and may even be projected several feet away from the bed.

On the other hand, when the aneurism ruptures internally into one of the cavities of the body, the symptoms are quite different. The patient feels something giving way within. Sudden collapse, asthmatic attack, and gradual exsanguination mark rupture into the pleura. Rupture into the pericardium is attended with intense pain, breathlessness, collapse, an anginal attack, and occasionally a convulsion. In rupture into the pericardium death is accelerated by cutting off the venous inflow, just as in simple pericardial effusion, only within a minute or two. Of course, under these circumstances no blood appears externally.

FIG. 302. Tracings of the outline of an aneurism of the innominate artery, showing the progress of its growth and the formation of secondary prominences upon its surface. Tracings made on Nov. 11, Nov. 23, and Dec. 2. One-third natural size.

Rupture of the aneurism into the pulmonary artery, vena cava, or right auricle or right ventricle sometimes occurs. The symptoms are usually sudden onset of dyspnœa, weakness, often collapse, and extreme cyanosis, which ends in death after a period varying from several hours to several months, the heart being unable to accommodate itself to the sudden changes in the distribution of blood.

However, in a large percentage of cases (863 cases = 47 per cent.- of Arnold's 1829 cases) death from aneurism is not due to rupture of the sac but "from pressure of the sac upon important nerves and blood-vessels, or from secondary changes which take place in these tissues and in other vital organs, as a direct or indirect result of such pressure." In 154 cases without rupture the causes of death were

Obstruction to air-passages	66
Exhaustion	50
Affections of lungs and pleura	28
Pericardial affections	8
Pressure on the vena cava superior	1
Collapse	1

Clotting within an Aneurism. The healing of an aneurism occurs by clotting within the sac. Since the latter is lined by arterial intima, there is under ordinary circumstances no more reason for clotting to take place there than elsewhere in the artery. As shown by Mall and Welch, arterial thrombosis occurs quite suddenly when the circulation is slowed and pulsation disappears, especially if there is some injury to the wall of the artery, and this is a most important factor in bringing about thrombosis within

an aneurism, though some fibrin ferment must be present. As a rule, in fusiform aneurisms the circulation is too strong and rapid for coagulation to set in, but in sacculated aneurisms a certain amount of fibrin collects along the wall. Each layer of fibrin serves as a filter for leucocytes, from which more fibrin ferment is generated and a second layer laid down, and so on until occasionally the entire aneurism may be filled spontaneously by a laminated clot.

Owing to the large area and great thickness of the fibrin deposited, and to the fact that the intimal endothelium is in most places still intact, there is little entrance of fibroblasts into the clot and little organization goes on. The aneurismal clot is, therefore, not converted into a solid mass of connective tissue as in endarteritis or thrombo-angeitis obliterans, but remains simply laminated fibrin. Deposits of calcium salts sometimes occur upon them, however, and tend to convert the obliterated aneurism into a solid tumor.

SYMPTOMS.

The signs and symptoms produced by aneurisms vary greatly, and depend upon the site at which they occur along the aorta, so that Broadbent has been "led to divide thoracic aneurisms into two classes,—namely, *aneurisms of physical signs* and *aneurisms of symptoms*, from the predominance of physical signs and symptoms respectively,—the former term applying to aneurisms of the ascending aorta and first part of the arch, the latter to aneurisms of the transverse and descending portions of the arch."

The *symptoms* produced by aneurisms arise secondarily as the result of pressure upon surrounding structures. *Shortness of breath* is frequent, resulting both from pressure on the trachea and bronchi and from concomitant disturbances in the circulation (embarrassment of heart action, stasis from pressure on veins). *Cough* is a common symptom, from pressure upon the recurrent laryngeal nerves as well as from bronchitis as a result of pressure (occasionally from tuberculosis). The pressure on the laryngeal nerve causes paralysis of the corresponding vocal cord and gives the cough a peculiar metallic quality known as "*the goose cough, brassy cough, stenotic cough, paretic cough*," etc. It is really the cough characteristic of paralysis of one vocal cord, and it is characteristic of aneurism only in so far as that the latter is the commonest circulatory disturbance in which laryngeal paralysis is a symptom.

Paroxysmal dyspnœa may occur, and especially in certain postures in which the trachea and bronchi are pressed upon by the aneurism. This "*asthma*" is the most common symptom of patients presenting themselves for treatment, and careless physicians often accept its presence as the final verdict, remaining oblivious to the true nature of the disease. Attacks of dyspnœa or suffocation very commonly come on during sleep when the laryngeal muscles relax and narrow the laryngeal slit. They occur especially when the patient falls into an unpropitious position, so that he soon finds it most convenient to sleep bolstered upright and leaning slightly forward with chin depressed. This position affords the maximum space about the air-passages with the minimum of tension upon them.

Not infrequently a small aneurism of the arch pointing backward and pressing upon the trachea or bronchi may cause actual *suffocation*, for which tracheotomy may be necessary. In some cases, however, the aneurism may be situated so low that it may be impossible to do the tracheotomy below the area compressed. The only possible means of relief is then to dissect or pull the aneurism away from the trachea, or to introduce a metal tube into the latter and thus hold the trachea open at the point compressed. This procedure is, of course, extremely difficult, and under all circumstances great dyspnœa from pressure on the trachea is in itself a dangerous symptom.

Pain is a common symptom in aneurism, and may be of three kinds:

1. *Angina pectoris*: reflex referred pain over the heart or down the arm. This is especially common in early aneurism at the beginning of the ascending aorta and from the sinus of Valsalva, and is probably due to changes in or pressure upon the aortic plexus. After these changes have been long established this pain may disappear (Osler).

2. *Sharply localized pain* may arise in or about the aneurism itself when its walls are pressed upon, or even spontaneously, and especially when it begins to erode the chest wall.

3. A second form of *referred pain* arises without reflex mechanism directly from pressure upon the intercostal nerves and those of the brachial plexus, especially in aneurisms of the transverse and descending aorta. The latter may give rise to pain in the back, shoulder-blades, and sides and also down the arm, and may for a while be mistaken for intercostal neuralgia or for the pain of pleurisy. Pain down the arm is especially common when the aneurism involves the innominate or subclavian arteries, particularly when the return of venous blood is interfered with by pressure on the veins.

Difficulty in swallowing and the feeling of a lump in the throat may result from pressure upon the œsophagus, especially when the aneurism is adherent to it or is infiltrating its walls. This is, of course, characteristic of aneurisms of the descending portions of the arch and to a less degree of the descending aorta. It is not at all a rare symptom, and yet is by no means as common as might be expected even when the aneurism is large.

PHYSICAL SIGNS.

1. The presence of a *visible mass* upon the chest wall or elsewhere showing a pulsation of an elevation which begins about 0.05–0.10 second later than the ventricular systole (or the first heart sound), and which on palpation is felt to be forcible and expansile in character (i.e., presses outward in all directions). The shock with the first sound is usually well felt and often accompanied by a thrill; and a diastolic shock accompanying the second sound is, when present, almost characteristic.[1] On auscultation there is usually a systolic murmur heard over the aneurism, and occasionally a diastolic murmur when the blood flows back into the aorta during diastole, especially through a narrow opening. This is, of course, most common and most marked when aortic insufficiency is present, either organic or relative, resulting from the general dilatation of the aorta.

Long before an aneurism perforates, and in many cases when it is not pointing outward but upward toward the episternal notch or clavicles,

[1] The writer has seen one case of hypernephroma of the thigh in which a diastolic shock was palpable, but this is rare even in the most vascular tumors, such as the vascular mediastinal sarcomata.

there may be seen a diffuse systolic lifting of the whole chest wall or of the parts above the tumor. The localized heaving is, of course, most marked when the aneurism is in the vicinity of cartilages or articulations in the chest wall and in younger individuals; while the most diffuse heaving occurs in the portions and persons where the ribs and sternum are most rigid. The impulse thrill and shocks, systolic and diastolic, are also frequently present when no heave or pulsation can be seen.

For discerning and timing slight pulsations the writer has frequently found it convenient to hold the index finger a few millimetres away from the chest wall, and watch for either a periodic narrowing of the slit between the finger and the chest or for a visible movement of the shadow cast by the finger upon the chest. For the latter purpose the light should strike as nearly as possible parallel to the chest so as to magnify the movement of the shadow (Fig. 303).

FIG. 303.—Method of inspecting for pulsations. *E*, eye of observer looking down from above. *E'*, eye of observer upon level with pulsating area. *f*, finger above pulsating area. *S* and *S'*, shadows thrown by the finger upon the pulsating mass. Solid and dotted lines represent the outlines of the pulsating mass, arrows indicate the extent of movement seen.

When aneurism is suspected, it is particularly important to examine the patient's back as well as to inspect carefully the front of the chest. This precaution was always particularly emphasized by one of the great teachers of medicine who also unintentionally illustrated its importance. On one occasion he and another professor, who was visiting the clinic, demonstrated to the junior students a case of suspected aneurism, but sent him to the wards for X-ray examination without having examined the back. The absolute diagnosis was at once made by the house officer, who in the routine examination discovered a well-marked pulsation at the back at the level of the third thoracic vertebra.

Dulness on percussion is of course present over an aneurism as over other tumors, the note being flat when the tumor is near the surface, slightly improved when it is deep. This improvement is often very slight in deeply situated aneurisms of the arch, and percussion, especially in the first right and left interspaces and over the manubrium, should be very carefully carried out when aneurism is suspected. The exact outlining of an aneurism by percussion may be very difficult. The area of dulness on even the lightest percussion may be considerably greater than that of the aneurism itself, just as is true of the cardiac dulness, see page 95. The uniform dilatation of the aortic arch (to about twice its normal diameter) which is so frequent in aortic insufficiency may give an area of dulness over and on both sides of the sternum which may lead to a diagnosis of aneurism (Fig. 311). The true nature of the condition can be shown only by the X-ray (Fig. 308).

In large and in deeply situated aneurisms direct percussion of the vertebral spines by Kouwy's method may show an unusual dulness over the corresponding area (especially between the third and the sixth thoracic spines) and may prove of assistance in establishing the diagnosis. It is, of course, of no value in those aneurisms

of the arch in which the trachea is interposed between vertebræ and the tumor. The heart is occasionally very much displaced by an aneurism which may itself come to occupy the usual site of the heart, so that on casual examination it may be mistaken for the latter. With careful auscultation, however, this error may be excluded.

Pressure upon the sympathetic on either side may give rise to inequality of the pupils, usually with a d i l a t a t i o n upon the affected side. In late stages, however, the sympathetic on that side may be completely destroyed and the pupil then becomes s m a l l e r on the affected side. The dilatation is best seen when there is moderate illumination, for in strong lights the reflex pupillar constriction may overcome the dilator action of the sympathetic.

Tracheal Tug.—W. S. Oliver, who described the sign in 1878, gave the following directions: "Place the patient in the erect position and direct him to close his mouth and elevate his chin to the fullest extent; then grasp the cricoid cartilage between the finger and thumb and use gentle upward pressure upon it; when if dilatation or aneurism exist the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand." This tracheal tug is no absolute sign of aortic dilatation. It is as readily produced when solid mediastinal tumors or enlarged bronchial glands adhere to both aorta and air-passages as from aneurism.

Sewall finds that in a large percentage of tuberculous individuals (91 out of 212—43 per cent.) a slight twitch of the trachea may be felt during inspiration, due to contraction of the accessory muscles, but this is not continuous, not synchronous with the pulse-beat, and should not be mistaken for the true tracheal tug. Moreover, in his large series of observations this tracheal twitch of non-aneurismal origin was always confined to inspiration. Sewall found it particularly common in cases of tuberculosis or old pleurisies in which there was adhesion to the left pleura. Mediastinal adhesions anchoring the aorta to the air-passages can produce it. Wenckebach has also called attention to the fact that it may occur in cases of enteroptosis, in which the heart is pulled downward with the liver, and the arch of the aorta thus made to pull upon the bronchi. (This is well illustrated by the patient mentioned on page 602.) The tracheal tug is most marked in inspiration.

Tracheal Percussion Shock (Smith).—H. L. Smith has found that if one lightly taps the chest wall (direct percussion) over an aneurismal area one feels a sudden increase in the impulse as soon as the aneurismal area is reached, a shock resembling "the sensation experienced by one when a rubber bag filled with water is simultaneously palpated and percussed" (semi-fluctuation). The fact that he has been able to elicit it in 62 per cent of his cases of which only 46 per cent gave a tracheal tug indicates the usefulness of the sign. In certain cases it is undoubtedly of considerable assistance.

THE PULSATION AND ARTERIAL PULSE IN ANEURISM.

The pulsation over the aneurism resembles the form of the arterial pulse except that owing to the elasticity of the sac the rise and fall are usually more gradual. It is sometimes of importance to determine whether the pulsation corresponds to an aortic aneurism or an aneurism of the ventricle. This requires the most carefully timed tracings simultaneously

from apex and tumor. If the pulsation in the aneurism begins .07–.09 second later than the apex beat, the aneurism may be assumed to arise from the aorta. If it arises from the ventricle, the two will, of course, be synchronous. On the other hand, as occurred in a case still under the writer's observation, the two pulsations may be absolutely synchronous and yet X-ray and other signs may show that the aneurism arises from the aorta.

Delay and Inequality of the Pulse.—Inequality of the pulse in cases of thoracic aneurism was recognized by Harvey, who stated very correctly that "the pulse in the corresponding arm was small in consequence of the greater portion of the blood being diverted into the tumor and so intercepted." The nature of this inequality was made the subject of a careful clinical and experimental study by Marey and François-Franck, who found that when an aneurism with elastic walls occurred along the aorta it served to damp the oscillations of pressure in the arteries nearest to it and thus to make the pulse smaller in these arteries. The pulse-wave thus became smaller and its onset less sudden, the upstroke becoming very oblique (pulsus tardus).

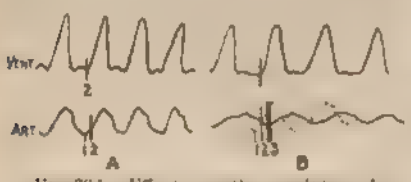

FIG. 304.—Effect upon the circulation of interposing A an inelastic and B an elastic bulb along the course of an artery in a model of the circulation. Modified from François-Franck. 1, 2, 3 represent successive time markings. *VKVT* pressure curve within the model of the ventricle, *AKT* pressure curve within the model artery. *A* normal artery. The elastic sac diminishes the size of the pulse, delays the upstroke, and delays the transmission of the pulse-wave.

Certain aneurisms, however, have no effect on the pulse, and it may be even larger upon the side of the aneurism than upon the unaffected side. Marey and François-Franck showed upon their models that if the sac was inelastic the pulse-wave was increased on the affected side, and its character became collapsing.

Owing to this damping of the pulse-wave, the maximal and minimal blood-pressures in the arteries nearest the aneurism tend to approach the mean pressure, so that the maximal pressure may be from 5 to 30 mm. lower than the maximal pressure in the opposite arm, though there may be a considerable difference in the size of the pulses without any marked difference in maximal or minimal pressures. The minimal pressure, being already nearer to the mean, is less affected than is the maximal, and often no difference can be noted. Changes in the size and in the quality of the pulse in the two radial arteries are much more marked to the palpating finger than to the instrument, for the slowed circulation may be compensated by a local vasoconstriction which causes a smaller pulse without appreciable change in the pulse-wave. The pulse is usually smaller and less sudden on the side nearest the aneurism, and hence often appears to be retarded when this is not actually the case, though in actual fact the onset of the pulse-wave is synchronous and only the summit of the pulse-wave is belated (François-Franck, Marey, v. Ziemmssen) (Fig. 283). François-Franck and Marey showed, however, that both in man and in the model, the presence of an elastic aneurismal sac along the aorta caused a general slowing in the transmission of the pulse-wave (apex—radial pulse interval = 0.2–0.22 second; costal of 0.12–0.14 second); in all the arteries, so that the pulse-wave in both radial arteries begins at exactly the same time. When the aneurism arises not from the aorta but upon the innominate or the subclavian artery, the result is different. The pulse-wave in the aorta now advances at the usual rate (apex—radial = 0.12–0.14 second), while the

In cases in which aortic insufficiency is present, especially with aneurisms of the ascending arch, this delay of the pulse-wave is not present.

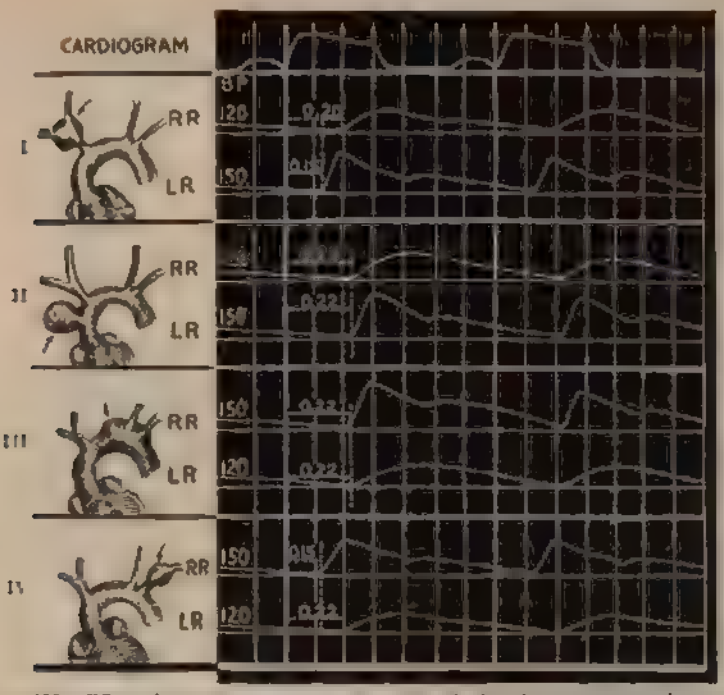

FIG. 305.—Effect of aneurism at various sites upon the blood pressure, rate of transmission and the form of the pulse-wave. Schematic. *Bp*, blood pressure in mm. Hg. *RR* pulse in right radial artery. *LR* pulse in left radial artery. I Aneurism upon innominate artery. II Aneurism upon the ascending aorta. III Aneurism upon the transverse portion of the arch of the aorta. IV Aneurism upon the left subclavian artery. The first column of figures indicates blood pressure in mm. Hg, the second column indicates the transmission time of the pulse-wave in seconds. The figures given are typical though the differences are larger than are usually encountered.

transmission in the artery from which the aneurism arises is slowed. The pulse-wave is therefore, definitely retarded (0.05 to 0.07 second later than in the other radial), in some cases even when there is no difference in the suddenness of the upstrokes. This difference in the time of the pulse-waves when present furnishes a means of differentiating between aneurism of the innominate or subclavian artery, and demonstrates that the aorta itself is not involved (François-Franck). This fact may be of great practical importance in determining the operative treatment, especially where the shadow cast by the aneurism lies close to that of the aorta. However, this delay in the pulse does not occur in all aneurisms, but only in those whose walls are elastic, the greater number. It may also disappear as clotting occurs along the walls of the aneurism and the elasticity of the sac is thus lessened.

FIG. 306.—Radial pulse tracings from the right and left radial arteries of a patient with aneurism of the first part of the arch of the aorta. The upstroke of the pulse wave in the right radial artery, which is nearer to the aneurism than the left, is more gradual than that in the latter.

In view of these facts, it is evident that the form of the pulse tracing is of no more value than the simple palpation of the pulse in the diagnosis of aneurism, and the only graphic method of practical importance is that of taking simultaneous tracings from both radial or both carotid arteries

in cases where it is necessary to determine whether an aneurism is confined to the innominate, subclavian, or carotid artery, or whether it also involves the aorta. These relations often cannot be shown by the X-ray and rest upon this differentiation alone.

X-RAY EXAMINATION.

Most of the things which are *inferred* from physical examination can be actually *seen* with the fluoroscope, and an exactness of diagnosis can be reached which is utterly impossible with the ordinary methods. In 104 cases of aneurism thus examined by Baetjer, the clinical diagnosis had been correctly made in 70 per cent., there had been tentative diagnosis of aneurism in 20 per cent., and *an unsuspected aneurism had been discovered with the X-ray in 10 per cent.* These findings are particularly important, since it is just in these early cases when the physical signs are still indefinite that treatment may be profitably instituted.

FIG. 307.—Radiograph of a patient with a large aneurism of ascending aorta and the arch, viewed from behind. (Kindness of Prof. C. M. Cooper.)

However, unless certain precautions are taken in the examination an aortic shadow may be seen which may be diagnosed an aneurism even though *none be present.* This rounded shadow is cast by the arch of the aorta just to the left of the sternum, and it may be specially marked if the aorta is somewhat tilted, as sometimes takes place in enteroptosis (Wenckebach). Holzknecht has shown, however, that this error will not be made if the patient is also turned so that the rays pass from left back to right front (Fig. 81). The normal aorta thus lies in a plane parallel to the rays and is seen as a narrow nearly vertical band, with the light spaces of anterior and posterior mediastinum in front and behind it. The uniformly dilated aorta appears as a wide but uniform band. The aneurism of the ascending aorta appears as a battledore or tennis racket with handle up, the aneurism of the arch as a racket with handle down. Aneurisms of the innominate are separated from the aortic shadow by a clear space which is bridged by the narrow shadow of the artery.

As Baetjer states, it is most important to examine the chest for malformations and for misplacement of the aorta which might be mistaken for aneurism. Persistence of the ductus arteriosus (Botalli) must also be considered in shadows near that of the descending arch. The shadow of enlarged mediastinal glands is usually speckled or blotchy with occasional lighter areas, rather than uniformly dark, while the edges of sarcomata and other solid tumors are often irregular and may fade away gradually

into the surrounding tissues. Moreover, unless there is a considerable degree of intrasaccular clotting, an aneurism will be seen to expand during systole and to contract in diastole,[1] whereas a solid tumor will at most rotate upon its axis.

CHARACTERISTIC FEATURES OF ANEURISMS AT DIFFERENT SITES.

Characteristic features of thoracic aneurisms are given in the table below. It must be borne in mind, however, that these represent the conditions only while the aneurisms remain relatively small, for with their further growth they may press upon other structures and so present the picture of an aneurism affecting a different part of the aorta. Large aneurisms also displace the heart, and may even occupy the usual position of the latter.

FIG. 308.—Radiograph of a patient with diffuse dilatation of the arch of the aorta. Kindness of Prof. C. M. Cooper. The figure also shows dilatation of the left ventricle and slight dilatation of the left auricle.

FIG. 309.—Diagram of the radiograph shown in Fig. 308. The broken lines indicate the normal outlines. *AO*, aorta, *OES*, œsophagus, *LV*, left ventricle.

Aneurism of the Heart.—*Symptoms.*—Indefinite signs of cardiac weakness. *Physical signs.*—Two points of maximal impulse over which tracings show exactly synchronous pulsations (this point is far from pathognomonic). Irregular outline of cardiac dulness (encapsulated pericarditis, pleurisy, and tumors must be excluded). Sometimes systolic and diastolic murmurs over heart and aneurism not present over aorta. *Pulse.*—Feeble but equal and not delayed. *X-ray.* Bulging of shadow of ventricle or auricle with enlargement of shadow synchronous with systole of corresponding chamber. *Rupture.*—Into pericardium. Death often from cardiac weakness or coronary sclerosis.

Aneurism of Coronary Arteries.—*Symptoms.*—No characteristic symptoms. Occasional cardiac pain. *Physical signs.*—Arteriosclerosis. No characteristic signs or even signs of illness. (Aneurism usually size of pigeon's egg.) *X-ray.* No abnormal shadows. *Rupture.* Into pericardium in 19 out of 21 cases. In one case into pulmonary artery.

Ascending Aorta; Intrapericardial (Aneurism of Symptoms). *Symptoms.*—Angina pectoris. Attacks of cardiac asthma. Precordial pains.

[1] Holzknecht particularly emphasizes the importance of using the lead diaphragm in examining the edges of the shadow for pulsation.

Pain down right or left arm. Shortness of breath. Symptoms of cardiac failure predominate. *Physical signs.*—Distention of veins of head, neck, upper chest, arms; œdema of these parts. Tracheal tug absent while aneurism is small. Pupils equal if aneurism is small. Aneurism usually small, situated in second and third right interspaces. Pulsation in second and third right interspaces. Often signs of aortic insufficiency. General œdema from aortic insufficiency. *Pulse.*—Delay of pulse-wave uniform. Pulses may be equal in both radials or may be smaller in either. *X-ray.*—Inverted racket-shaped shadow in left post. to right ant. illumination. Arch of aorta clear. *Rupture.*—Into pericardial cavity. Pulmonary artery. Right auricle. Superior vena cava. Œsophagus. Left auricle. Right ventricle. Left lung. Right lung. *Other causes of death.* Dyspnœa. Exhaustion. Hydrothorax. Hydropericardium. Bronchitis and pneumonia. Pulmonary infarction. Suffocation.

Aneurism of the Ascending Aorta between Pericardium and Innominate Artery (Aneurism of Physical Signs). *Symptoms.*—Slight dyspnœa. Pain when aneurism presses on or erodes chest wall. Often an accidental finding. *Physical signs.* Flushed face with dilated veins; sometimes œdema. Dilated veins of arms. Pulsation in second, third, and fourth right interspaces (occasionally shifting). Dulness to the right of the sternum, not over the manubrium. Systolic, sometimes diastolic murmur, thrill and diastolic shock over aneurism. Tracheal tug, if aneurism is large. *Pulse.*—Uniform delay of pulse, both sides synchronous. Right radial usually smaller than left. *X-ray.*—To right of sternum in second to fourth interspaces, best made out in post.-ant. or right post. to left ant. illumination. In left post. to right ant. illumination inverted racket-shaped shadow. *Complications.*—Often aortic insufficiency. Bronchitis. Tuberculosis of right lung. Hemorrhage. Right hydrothorax. *Rupture.* Into pericardium. Right pleural cavity. Right bronchus. Right auricle. Superior vena cava.

Aneurism of the Innominate Artery. *Symptoms.*—Like those of aneurism of arch except that there is no dysphagia in small aneurisms. Pain and numbness down right arm and to right shoulder. *Physical signs.*—Dilated veins and swelling over right arm and right side of face. Dulness extends out under right clavicle. Pulsating tumor may be felt under the right clavicle. Paralysis of right vocal cord. Right pupil in early stages larger than left. *Pulse.* Right radial pulse smaller and definitely later than left. *X-ray.* V-shaped shadow is seen upon the left arm of the V which the shadow of the innominate artery makes with that of the aorta on left post. to right ant. illumination. *Complications.* Right-sided bronchitis. Bronchopneumonia. Tuberculosis. Hydrothorax. *Rupture.* Usually points upward and outward toward the clavicle, but may point downward to pleura or bronchi.

Aneurism of the Arch of the Aorta (Aneurism of Symptoms). *Symptoms.* Change in voice, especially high notes. Brassy cough. Difficulty in swallowing. Pain in throat. Dyspnœa, sometimes amounting to suffocation. *Physical signs.* Inequality of pupils. Usually dilatation of left pupil. Dilated veins, flush, and sometimes swelling over left side of face, chest, and left arm, or changes bilateral. Tracheal tug early. Pulsation palpable in suprasternal notch. Pulsation in suprasternal and supraclavic-

ular fossæ. Lifting of manubrium; later perforation of manubrium or sternoclavicular articulation. Palpable heaving, systolic and diastolic shocks, and often thrill over manubrium. Heart sounds: usually systolic murmur and sometimes diastolic murmur over the tumor. In aneurism beyond the innominate, the systolic murmur may be heard in the left carotid and brachial but not in the right. Bronchoscopy may show tumor perforating bronchus. *X-ray*.—Shadow racket shaped, especially seen in left post. to right ant. illumination. Post.-ant. or ant.-post. illumination seen as massive shadow above that of the heart. *Complications*.—Bronchitis. Tuberculosis. Suffocation (asphyxia). Inanition. *Rupture*.—Externally (anteriorly through manubrium or above clavicle). Into left bronchus. Trachea. Œsophagus. Lungs and pleural cavity, pericardium, mediastinum. Pulmonary artery. *Other causes of death*. Exhaustion. Pericarditis. Collapse. Suffocation. Œdema of larynx. Pneumonia. Tuberculosis.

Aneurism of the Descending Aorta.[1]—*Symptoms*.—Lancinating and boring pains in back, left shoulder, left side, and left side of abdomen. Stiffness of back. Shortness of breath. When the aneurism is near the diaphragm, abdominal pains may be present and the condition may be considered to be abdominal. *Physical signs*.—Visible pulsation just to left of spinal column. Dulness on percussion. Heart sounds and corresponding shocks over aneurism and tenderness over corresponding spines. Areas of hyperæsthesia or analgesia in corresponding spinal segments. *Pulse*. Pulses synchronous; smaller and more gradual in left than right. *X-ray*.—ç-shaped shadow to left of sternum, especially in right posterior to left anterior illumination. *Complications*. Left-sided bronchitis, bronchopneumonia, tuberculosis, hydrothorax, paraplegia from erosion of vertebræ. *Rupture*.—Backward and externally, into œsophagus, left pleural cavity, right pleural cavity, bronchi and lungs, pulmonary artery. *Other causes of death*.—Pressure on trachea and bronchi, exhaustion, pneumonia, and tuberculosis.

The following histories illustrate typical cases of aneurism.

ASCENDING ARCH ABOVE PERICARDIUM (ANEURISM OF PHYSICAL SIGNS).

D. N. L., aged 45, married. Except for a well-compensated aortic insufficiency for the past eight years, with slight shortness of breath, he has been quite healthy. In November, 1903, his aneurism was discovered accidentally by his brother, who is a physician.

Examination by Dr. Osler revealed a well-nourished man who does not appear ill. Face a little congested; veins of neck and arms full; pulse 48 per minute; both apparently synchronous; a little larger on left than on right; maximal pressure: left arm 140, right arm 125. There is no tracheal tug. Over the thorax a wavy impulse is seen in all the right interspaces above the liver, and an area of dulness as outlined in Fig. 310, A. Relative cardiac dulness in fourth interspace extends 10.5 cm. to left and 15 cm. to right of midline. Over the aneurismal area there is a marked systolic thrill and murmur over the heart, a systolic and diastolic murmur.

[1] In 120 cases of aneurism of the descending aorta collected from the literature Milanoff found pain in 72, dysphagia in 20, hæmatemesis 13, hæmoptysis 21, left-sided pleural effusion in a few cases. Andral found only 8 cases of paraplegia from aneurism in the literature. The duration is often from 10 to 15 years, longer than that of aneurisms elsewhere.

The condition is very well discussed in English by Osler and more recently by Hewlett and Clark, who give excellent monographs and a very useful summary of the literature.

X-ray examination by Dr Baetjer showed the pulsating shadow of an aneurism of size corresponding to the area obtained on percussion, arising from the ascending arch of the aorta

Patient left the hospital, and one month later died without warning while asleep. The only signs of the approaching end were fifteen minutes of stertorous breathing Autopsy revealed a large aneurism corresponding to that diagnosed clinically, as well as arteriosclerosis and aortic insufficiency. There is no note of rupture of the aneurism.

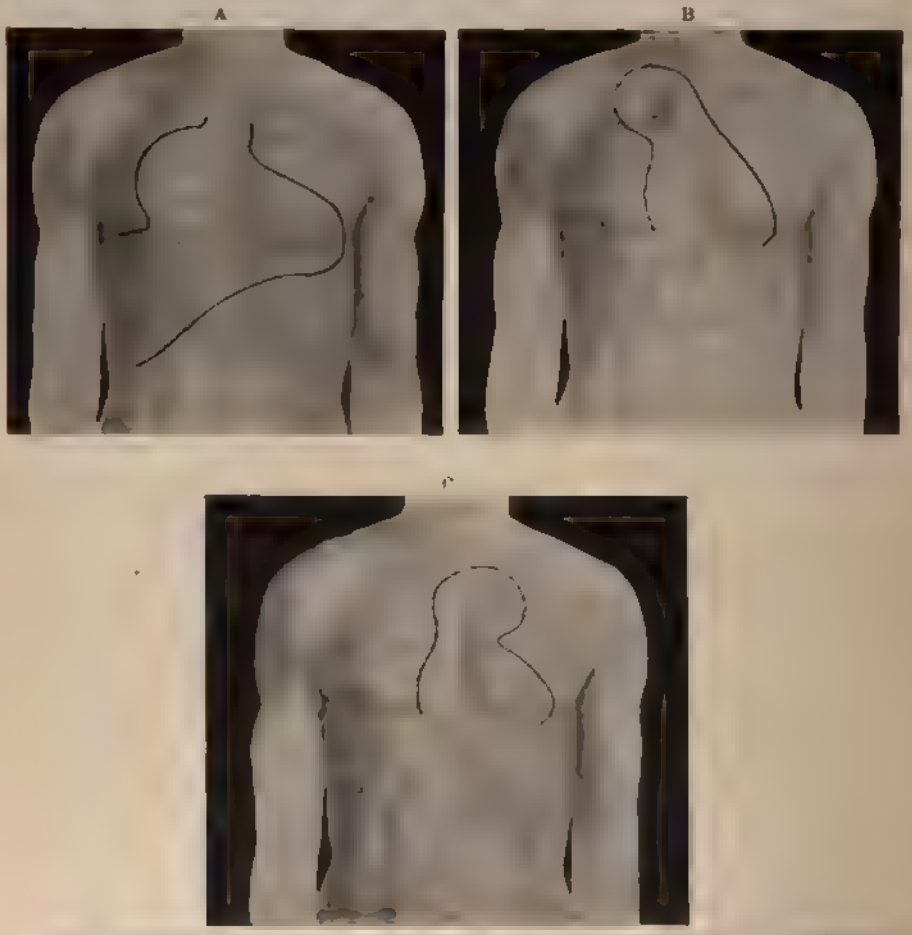

FIG 310 Cardiac dulness in cases of aneurism. A, Ascending aorta D, N L.N. B Subclavian artery (J B the shaded area indicates the tumor, the curve indicates the pulsation C, Second part of the transverse portion of the arch (Ky.

ANEURISM OF INNOMINATE ARTERY.

J B , aged 44. Had syphilis 13 years ago, otherwise healthy.

Two years ago complained of aching in right shoulder and right side of neck down arm to hand No pain in chest There were swelling of feet and ankles and shortness of breath About this time he began to have troublesome paroxysms of coughing In May, 1903, noticed that his voice was 'cracked' In August, 1903, he noticed a pulsating swelling above the right clavicle This was diagnosed as aneurism of the innominate, and the right carotid was ligated above the tumor. The subclavian could not be ligated, as the patient took chloroform

badly. After the ligation the tumor rapidly increased in size. When he entered the Johns Hopkins Hospital two months later, his voice was husky, the right pupil was larger than the left (irritation of the right sympathetic ganglia), and a large rounded pulsating tumor was seen occupying the position of the manubrium and extending out along the right clavicle (Fig. 310, A, B). The prominence of this tumor is shown in Fig. 302. Over the tumor a well-marked systolic and diastolic shock may be felt. The two heart sounds are heard over the tumor. There is some resonance on percussion between the tumor and the heart. The area of cardiac dulness is not enlarged, but a soft diastolic murmur is heard at apex and base. The right radial pulse is smaller than the left and a little delayed. The blood-pressure varied, at first being 160 in left arm, 140 in the right brachial, later reaching 150 in right and 130 in left.

Patient was seen by Dr. Finney, but wiring and other operative procedures were considered impracticable. He was kept at rest in bed on restricted diet, but nevertheless the aneurism grew rapidly, as shown by the successive elevations in Fig. 302.

On Dec. 2, at 4.00 P.M., he felt a severe throbbing in the aneurism, and a couple of smaller bulgings (Fig. 302) appeared upon its surface, which had not been present the day before, and the whole aneurism appeared to be definitely larger. The patient insisted upon leaving the hospital at once to return to his home in South Carolina.

Aneurism of the First Part of the Transverse Arch of the Aorta.

J. D., an unmarried sailor, aged 37, entered the Johns Hopkins Hospital on Jan. 5, 1900, complaining of pain in the chest and inability to sleep. He had had no infectious diseases except gonorrhœa, denied syphilis, and gave no history of secondaries. Except for occasional sprees, he uses alcohol in moderation. As a sailor he has always done heavy work.

He was well until about four months before admission, when he had an attack of "heavy pressure" and tightness across his chest at night, and some weeks later stinging pains in his chest 3–4 cm. above the xiphoid process, which seemed to radiate to both sides of the chest and to pass through to the back. The pressure kept him from sleeping.

Upon examination the veins upon the left side of the neck were found to be dilated, and the veins were much more prominent than on the right. There was no tracheal tug. There was an area of dulness behind the medial end of the right clavicle, the manubrium, and the medial half of the left clavicle, which was continuous with the upper border of cardiac dulness. The left radial pulse was somewhat smaller than the right and seemed to be a trifle retarded. Maximal pressure in the right radial ranged from 115 to 130 mm. Hg, in the left from 85 to 110 mm. Hg.

Fluoroscopic examination by Dr. Baetjer showed a pulsating tumor about the size of a hen's egg projecting almost entirely to the left of the sternum opposite the first and second interspaces (transverse portion of the arch of the aorta).

The patient was discharged somewhat improved after a short sojourn in the hospital.

Aneurism of the Left Side of the Transverse Arch.

H. D. K., brush-maker, aged 49, admitted to the surgical service of the Johns Hopkins Hospital on March 14, 1908, complaining of aneurism. Family history was negative. He had been perfectly healthy all his life except for an attack of pleurisy six years before admission, an attack of gonorrhœa at 19, and a chancroid not followed by secondaries at 25.

Two weeks before admission he felt a burning pain in the left chest and had some shortness of breath, both of which have become worse since then. He sometimes is awakened with shortness of breath and precordial pain. For the past year his voice has been husky, a condition which set in suddenly after violent exertion while splitting wood.

He is a well-nourished man of rather anxious expression. The pupils are equal and react to light and accommodation. There is a well-marked tracheal tug. Chest expansion is slight on respiration. There is a definite pulsation over the first left interspace and sternoclavicular junction, in which area the shocks accompanying the two sounds are readily palpable.

Note by Dr. Hoops.—Heart: Maximal impulse in fifth left interspace 11 cm. from midsternal line; dulness extends to this point on left, reaches above to middle of third rib, and on right 2.5 cm. in third left interspace. Cardiohepatic angle is normal. There is dulness behind the manubrium, extending to left as per diagram, 8.5 cm.

in first interspace and below to second interspace, on the right to just beyond the sternal margin. On palpation over the dull area there is a strong lifting pulsation, maximal at a point 3.5 cm. to left and definitely expansile. No thrill felt. Diastolic shock well marked. At apex and inward toward the base there is a very short systolic murmur, which is not transmitted beyond the border of the heart. First sound is rather tapping at apex. Along the left sternal border this murmur increases in intensity and is maximal over the mass above the heart at the point of greatest pulsation, where there is a well-marked systolic bruit followed by a ringing second sound. A very faint systolic murmur is heard at the aortic ring, and the second sound is clear. No diastolic murmur. The second pulmonic is louder than the second aortic. The pulse is of good volume, regular, rather high tension, and not collapsing. Vessel wall definitely thickened. Some cyanosis of finger-tips and lips. The volume of the pulse on the right side is decidedly larger than on the left. There is a circumscribed area of dulness in the left interscapular region in which percussion note has a peculiar wooden tympany like that over consolidated lung. Over this area the breath sounds are rather more intense than over the rest of the lung but they are not tubular in character.

On January 20 the aneurism was wired, under Schleich solution anæsthesia, by Dr. Finney, with twelve feet of silver-copper wire, through which a 10-M.A. current was passed (Moore-Corradi method). Clotting took place promptly. The patient stood the operation well. The patient was considerably relieved as regards pain, but the pulsation soon returned.

Aneurism of the Descending Aorta.

Notes of the following case are taken from the records of the Johns Hopkins Hospital.

Ch. L., colored laborer, aged 48, was first admitted to the Johns Hopkins Hospital on October 14, 1898, complaining of pain in the back, left side and abdomen.

His family history was negative. He had always been healthy, but had measles, whooping-cough, tertian malaria, and at 17 had syphilis which was not adequately treated. He has done a great deal of hard work on a farm, has drunk a great deal of whiskey, and smoked heavily.

His present trouble began suddenly about four years ago, when he was seized with a severe pain in the lower left abdomen. This lasted a couple of weeks. It was always relieved when his thighs were flexed upon the abdomen, and was always increased after exposure to bad weather. Four years after this a pain in the left side of the back appeared, which has gradually increased. This also is relieved by flexing the thighs. For the past six years he has passed blood in the stools during periods when the pain in the left flank was worse.

A note by Dr. Futcher at that time states, that "the patient was found lying in bed on the left side with the knees flexed. Pupils are of normal size, equal and react to light and accommodation. The lung expansion and vocal fremitus are diminished over the entire left lung, and the breath sounds were exaggerated in front and in the axilla. There were a few moist rales in the third and fourth left interspaces. Behind, the breath sounds are very indistinct below the angle of the scapula. The percussion note was found to be impaired over the entire left front as far down as the fifth rib and over the entire left back, being flat below the angle of the scapula.

Heart.—The maximal impulse was seen in the fifth interspace 7.5 cm. from the mid-line, but dulness extended 3 cm. to the left of this point. Both the first and second sounds were reduplicated.

The liver was slightly enlarged, the spleen just palpable. There were no masses nor areas of tenderness in the left flank to account for the pain.

On Oct. 21 Dr. Futcher noted a definite heaving of the entire body of the sternum and a well-marked systolic retraction in the eighth, ninth, and tenth left interspaces behind. A tracheal tug was present, but the vocal cords were not paralyzed. The pulse was equal on the two sides.

In spite of rest, restricted diet, and potassium iodide and repeated gelatin injections his pain in the back gradually became worse, compelling him to seek relief by leaning over the back of the chair. It became so severe that it was not relieved by .30 mg. (gr. ss) of morphine. However later in his stay his condition gradually became better and the pain became a little less frequent and less intense.

There was very little change in his condition between that time and March, 1902 when for the first time there was noted a definite systolic pulsation in the left interscapular region which gradually increased until it involved three ribs and interspaces. His condition gradually became worse. Respiratory movement almost entirely disappeared upon the left side and a scoliosis developed with concavity toward the right. The area of cardiac dulness increased to the right, where pulsation was particularly well marked and a superficial scratchy systolic murmur was heard over the precordium. His pain became so intense that he could but rarely lie down. An area of absolute analgesia developed in the sixth and seventh left interspaces, impaired sensibility to heat, cold, and pain being found in the fifth interspace as well. About this time he began to feel pain on swallowing, referred to the middle of the sternum.

During the night of Oct. 25 he complained of pain and intense shortness of breath, and suddenly vomited about 50 c.c. of bright red blood. Ten minutes later he vomited 25 c.c. more blood. He was quieted with morphine during the night, but in the early afternoon of the twenty-fifth he vomited about 500 c.c. of blood within three minutes, became pulseless, and died.

At autopsy the heart was found to be displaced to the right (extending 8 cm. to the right of the midline) by a tremendous aneurismal sac 18 × 14 × 9 cm. This sac was fusiform with sacculations at its upper and lower ends. It arose from the descending part of the arch and the descending aorta itself and pointed backward, eroding the bodies of all the thoracic vertebrae from the fifth to the tenth, as well as the seventh, eighth, and ninth ribs. The erosion of the intervertebral disks was much less marked. The aneurismal sac also compressed the œsophagus at the level of the bronchial bifurcation, where it eroded through the œsophageal wall, making an opening 2.5 cm. in diameter. "The edges of this aperture were rugged and necrotic, the tissue about it dark gray-green in color."

The aneurismal sac was partly filled by a large lamellated clot.

The ascending aorta was dilated and atheromatous, the descending aorta below the aneurism likewise.

The heart was much enlarged; the walls hypertrophic; the valves normal.

There were many pericardial adhesions, especially firm over the left auricle and the coronary veins, and there were tortuous patches over both ventricles.

The stomach contained a litre of clotted blood. Other organs normal.

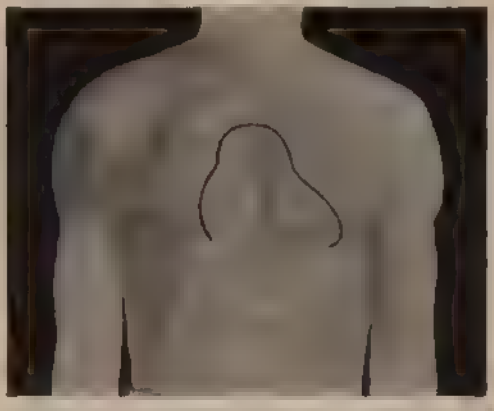

FIG. 311.—Area of cardiac dulness in a patient (L. D.) with dilated arch of the aorta.

Simple Dilatation of the Arch

L. D., gardener, aged 55, native of Ireland, came to the Johns Hopkins Hospital Dispensary on July 13, 1909, complaining of pain on swallowing and trouble in passing water.

The family history was negative. The patient had smallpox at 18, gonorrhœa at 35 and again at 48, and a chancre at 35 followed by definite secondary manifestations for which he had been given medicine by mouth. He has drunk whiskey in excess and has done a good deal of heavy work.

He was perfectly healthy until the past ten days, since when he feels food passing down his œsophagus and has a little pain which is referred to the level of the cardia. He vomits immediately after eating, but can swallow liquids without difficulty.

Examination reveals a fairly nourished man of ruddy complexion with some dilated venules. The left pupil is somewhat larger than the right, though both react to light and accommodation. There is a slight but definite tracheal tug, no tracheal percussion shock. There is no glandular enlargement. The lungs are clear except for a few widely scattered piping rales.

The heart is not enlarged; apex in fifth left interspace 10 cm from the midline. Dulness extends 1.5 cm to the right. The relative dulness is continuous above with a strip 3.5 cm upon either side of the sternum, over which the percussion note is very slightly impaired. This area extends up as far as the upper border of the second rib and is shown by the fluoroscope to correspond with a uniform shadow of the dilated aortic arch. The heart sounds are clear, the second aortic distinct, no diastolic murmur present. Pulse is of good volume, not collapsing.

Abdomen shows no masses, no visible peristalsis. There are no tenderness and no abnormalities palpable. The stomach tube is passed into the stomach without difficulty and a very small amount of clear fluid free from HCl, obtained, lavage fluid clear. Desmoid test negative. He was given alkaline gentian tincture + strychnine (1 mg. – 1/30 gr.) before meals, under which treatment his symptoms rapidly diminished.

Diagnosis. Chronic alcoholic gastritis, anacidity, dilatation of the aortic arch.

Aneurism of the Abdominal Aorta

M. P., machine agent, aged 30, was first admitted to the Johns Hopkins Hospital on Oct. 31, 1899, complaining of kidney and stomach trouble. The family history was negative.

The patient had had measles, chicken-pox, mumps, and whooping-cough, typhoid fever at 23, followed by pain in the ankles and knees. He had gonorrhœa two years before admission (about one year before the onset of the present trouble) but denies lues. He worked on a farm until his attack of typhoid fever, since when he has not been strong. He does not drink nor smoke and is a hearty eater.

The present illness began six months before admission, with some soreness and pain in the abdomen, which had no relation to the taking of food except that it was more intense after a large meal. He vomited occasionally but rarely. The pain was at first a sharp throbbing pain in the left side. It was so severe as to cause him to remain in bed for a period of three months, during which he had to be given morphine. After the three months' sojourn in bed the pain became less, and he was almost free from symptoms for about six weeks, when he was taken with a sudden sharp cutting pain in the right side just under the ribs, running around toward the right and down toward the testicle.

On examination he was found to be a well-nourished man of rather sallow color. There was no glandular enlargement. The lungs were clear on auscultation and percussion. The heart was not enlarged, the heart sounds clear.

In the abdomen there was a very well-marked pulsation visible in the epigastrium. There was dull tympany over this area, and inflation of the stomach showed that the pulsating mass was covered by the latter. There was considerable tenderness over the pulsating area. Dr. Osler was able to make out a definite soft systolic murmur, and on Nov. 5 with the deepest possible palpation could make out a definite mass with expansile pulsation. The case was diagnosed as abdominal aneurism and wiring was advised. This was performed by Dr. Finney two months later.

Note by Dr. Finney.—An incision was made 2.5 cm. below the xiphoid. The stomach was retracted downward, the lesser peritoneum was opened. The pancreas covered the lower surface of the tumor. Attempt was made to dissect the pancreas. This was abandoned on account of profuse and persistent hemorrhage. The incision was then enlarged upward, the edge of the liver elevated, and the tumor exposed above and to the right of the pancreas. A needle was inserted at this point to a depth of 3–4 cm., and 8–9 feet of silver and copper alloy wire introduced. Ten milliamperes of current were passed for one hour. The needle was withdrawn, the wire cut close to the aneurismal sac and turned in with a clamp. No bleeding. One or two bleeding points about the pancreas were tied with fine silk. The incision was closed. The patient made an uneventful recovery and experienced considerable relief from pain, so that nine months later it gave him little trouble, though the aneurismal pulsation was still expansile. There was now a loud systolic murmur over the mass.

The pain, however, gradually returned and never left him. It was so severe that he was a frequent visitor at the hospital and was compelled to use a good deal of morphine. He was admitted to the writer's ward in Jan. 1901, somewhat worse than at any time previously. The aneurismal mass now extended from the ensiform to within 3

centimetres of the umbilicus. Its surface was smooth and no areas of bulging could be made out.

On Jan. 7 the leucocytes were 5000, the haemoglobin 90 per cent. He was quite well (when given morphine) until the night of Jan. 21, when he had a sudden attack of most intense pain in the lower back and abdomen "causing him to cry out and toss about, arching his back and stiffening all his muscles in his attempts to bear the pain in silence." There was no objective change in the abdomen, but the tenderness over the aneurism was more marked than before.

The next day he had several attacks of pain and vomiting. At 4.00 P.M. the vomiting was very severe and was accompanied by intense pain and sudden collapse. His color became a ghastly pallor. He became almost pulseless before any one could reach him. He was still conscious and complained of great pain in the back and right side of the abdomen, to relieve which 15 Gm. (gr. ssss) of morphine were necessary, given within an hour. At 5.45 P.M. a small saline infusion was given and caused the pulse to improve slightly. Strychnine, 2 mg. (1/30 gr.), + digitalin (German), .15 mg. (1/400 gr.), had no effect. The maximal blood-pressure before the collapse was 130 mm. Hg, after it was 70 mm. Hg.

The next day there was dulness throughout the right flank extending up to liver dulness (due to outpouring of blood into the peritoneal cavity). The systolic murmur over the tumor disappeared, but the aneurismal mass still pulsated. The haemoglobin was found to have fallen to 55 per cent.; the leucocytes rose to 17,500.

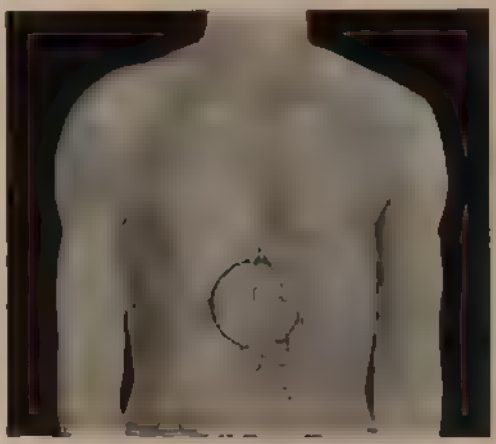

Fig. 312.—Tumor and pulsation in a case of patient (M. P.) with aneurism of the abdominal aorta.

During the next few days the patient's condition seemed to improve. The pulse became stronger, the maximal blood-pressure rose to 120 mm. Hg. However, his kidneys absolutely ceased secreting. He did not void at all spontaneously, and 50 c.c. of clear reddish liquid of neutral reaction and with a specific gravity of 1030, was all that could be obtained on catheterization on the evening of Jan. 24. It contained a large amount of albumen, no sugar, no casts, a few red blood-corpuscles, and a large number of pus-cells. This was the last urine obtainable even by catheter.

From this time on the patient's condition became worse. He complained of sudden shocks like electric shocks through his nervous system, to which he responded by sudden single twitches. He had no general convulsions. His mind remained perfectly clear, his pulse good until the morning of Jan. 26 (five days after the rupture), when his pulse gradually became weaker, he lapsed into unconsciousness, and died at 10.30 P.M.

Autopsy confirmed the clinical diagnosis showing a large saccular aneurism of the abdominal aorta which had ruptured into the retroperitoneal tissue and lesser peritoneal cavity, causing infarction of the left kidney and obliteration of the vessels to the right. There was thrombosis of adrenal vessels and a tremendous hemorrhage into the greater peritoneal cavity as well. This rupture had evidently occurred at the time of the collapse on Jan. 22. There was an island of clot within the coil of silver wire within the sac but a wide free blood channel between this clot and the aneurismal wall, so that the clot had not strengthened the latter in the least.

It is probable that during the months following the wiring, while he was free from pain, the clot filled the entire aneurismal sac, and that the eddy currents dissected it free from the aneurismal wall about the time that the pains returned.

DIAGNOSIS.

The diagnosis of thoracic aneurism is, as a rule, easy, especially with the aid of the X-ray. Most of the conditions with which it can be confused have been mentioned above. The most important of these are simple dilatation of the aorta, mediastinal tumors, pulsating empyema or encapsulated pericarditis, and enlarged mediastinal or branchial glands. Any of these may cause the dulness, the tracheal tug, the inequality of pupils and pulse. The systolic thrill and murmur may also be communicated by a very solid tumor or may arise in a very vascular sarcoma, aberrant thyroid with stroma, or metastasis from a medullary carcinoma or hypernephroma. A diastolic shock is scarcely ever felt over even the most vascular tumors, but is, of course, well marked over a dilated aorta. The presence of a forcible *expansile* pulsation in the interspaces is sufficient to exclude tumors; but in the first and second interspaces when there is no actual bulging it may be due to a simple dilatation of the aorta. The tracheal tug may further be due simply to displacement of the heart or aortic arch or to enteroptosis, while the inequality of the pulse may arise from the presence of adhesions or arteriosclerotic plaques about the origin of the subclavian arteries.

FIG. 313.—Tortuous subclavian artery, simulating a small aneurism. The tumor which it formed above the clavicle is indicated by the shading.

The absolute diagnosis can almost always be made by *X-ray examination*, but even then a tumor may be encountered whose shadow shows no expansile pulsation and whose nature remains in doubt. The homogeneous shadow, with its regular spherical or oval form and its connection with the aorta, is usually evidence of aneurismal nature.

In doubtful cases the greatest care is necessary, for the physician should always bear in mind that the earlier the aneurism can be treated the greater the chance of cure, and this stage of hope is usually the stage in which the physical signs are still far from definite.

Occasionally a tortuous carotid or subclavian artery presenting its convexity in the supraclavicular fossa may simulate an aneurism, so that, as in the case seen in Fig. 313, it is necessary to outline the supposed aneurism with the tip of the little finger. In this case, which had once been diagnosed aneurism, it was possible to reach below the convexity and to feel the outline of a narrow but tortuous subclavian artery. Of course, the X-ray examination in such a case would at once clear up the diagnosis, even if the outline of the artery could not be felt.

Another condition which may simulate aneurism of the subclavian artery is a dilated jugular bulb, which appears as a pulsating sac above the clavicle. This is especially marked when tricuspid insufficiency is present. In such cases the arterial blood-pressure may be low and the arterial pulses weak, nevertheless, the pulsation is so feeble and the connection with the dilated veins so evident that it should never be mistaken for an aneurism.

DISSECTING ANEURISM

Shakelton in 1822 gave the first descriptions of dissecting aneurisms, which were soon confirmed by Hope (1833) and Henderson (1843). In this condition the coats of the aorta are split longitudinally into two sleeves, —an outer, originally formed by the adventitia, and later lined by new-formed intimal endothelium; and an inner sleeve representing the original tube of the aorta, composed of the original intima and media, and later also covered with new-formed endothelium.

PATHOLOGY.

The condition is not an extremely rare one, so that Bostroem in 1887 was able to collect reports of 177 cases. It usually arises in the aorta, especially at the beginning of the descending arch, and not infrequently is formed as the continuation of a simple aneurism of the arch. From this region it commonly extends along the whole length of the aorta to

FIG. 314.—Dissecting aneurisms. A, Specimen of a dissecting aneurism (partial clot formation) in a man with only two aortic cusps. From the Army Medical Museum, Washington, D.C. B, Dissecting aneurism of the arch, involving the arch and the descending aorta. After Macfallum, kindness of the Johns Hopkins Hospital Bulletin.

the bifurcation, the arteries sometimes arising from the inner, sometimes from the outer tube. Occasionally the split occurs between the layers of the media, so that both sleeves have a wall of media. Very infrequently the outer tube ruptures into the inner tube near its lower end, so that the blood passes back into the latter.

The most satisfactory explanation for this remarkable lesion seems to be the following (v. Müller, Flockemann, Schede): As long as the lumen of the artery is uniform, the blood exerts only a lateral pressure upon the

arterial walls, which acts "across the grain" of the arterial coats. However, when calcified plaques project into the lumen, these tend to impede the blood-current so that the longitudinal pressure of the latter acts as well As Bostroem has shown, the resultant force acts in a parabola pointing outward and downward When this is acting upon an area where the media is thinned or absent, it tends not only to split the coats "with the grain" but also to push the adventitia outward. The wall gives way, the split lengthens, and the outer sleeve is formed. Whether or not the aorta then ruptures depends upon the ability of the adventitia alone to withstand the blood-pressure.

The coagulation of the blood within the sac depends upon the formation of fibrin ferment in the tissues of the adventitia and the rapidity of the blood-flow within the new-formed sac. It is quite frequent for extensive and even total coagulation of the contents to take palce.

SYMPTOMS AND SIGNS.

A considerable proportion of the cases of dissecting aneurisms give no outward manifestation during life and are accidental findings at autopsy. A large number give the usual signs of ordinary aneurism, especially when they arise as a continuation of the latter. This is well exemplified by the following case, which was under the writer's observation during his last admission to the hospital (The pathological findings and clinical notes are taken from the report of Professor MacCallum.)

L R, negro, aged 30 had been treated in the Johns Hopkins Hospital one year previous to his final admission, at which time the diagnosis of aneurism of the aorta had been made At the final admission the patient was found to be suffering from an arthritis with symptoms of general fever, sweating, etc The heart was enlarged, dulness extending 13.5 cm to left and 3 cm to right of midline There was visible impulse and heaving in second, third, fourth, fifth and sixth interspaces, far out in first and second left interspaces Heart sounds were clear dull and ringing, second sound followed by soft diastolic murmur in third left interspace, not heard in neck Patient died in delirium with high fever

Autopsy showed general streptococcus septicæmia hemorrhagic nephritis, acute purulent arthritis, obliterative pericarditis aneurism of ascending aorta dissecting aneurism of the descending aorta The aortic orifice is not dilated (8 cm in circumference) A large aneurismal sac (7 cm in diameter) lies behind the pulmonary artery, it extends upward in the aorta to the arch, beyond which the tube becomes double, the inner tube (the original lumen of the aorta) having for its walls the original intima plus media the outer tube media plus new-formed endothelium Numerous trabeculæ jut out transversely into its lumen Some of the intercostal vessels arise from the new, some from the old lumen The left renal artery arises from the old lumen, the right has been torn and arises from the outer At the lower end above the bifurcation the outer tube has ruptured back into the original lumen

Another type is exemplified by a case under the care of Professor Halsted, also reported by MacCallum.

Patient, aged 60, subject to mental disturbance and epileptiform attacks complained on May 28 of intense pain over the whole body which he could not locate. On May 28 his abdomen was much distended and he was jaundiced There was pain in the region of the appendix. His temperature was 100° Leucocytes 20 000 Exploratory laparotomy showed an extremely distended colon which was relieved by colostomy Patient died the next day.

Autopsy showed a dissecting aneurism along the whole aorta, splitting the media. The outer sleeve of the descending arch perforated into the posterior mediastinum, giving rise to a tremenduous hæmatoma which distended that space down to the diaphragm. The rupture had evidently given rise to the pain; the disintegration of red corpuscles in the clot had caused the hæmatogenous jaundice. Both these phenomena are common in cases of this type.

ANEURISM OF THE PULMONARY ARTERY.

Aneurisms of the pulmonary artery are very rare as compared with those of the aorta. Henschen (1906) has recently summed up the reported cases. In contrast to aortic aneurism, he finds that there is no close relationship to hard work; 18 out of 34 cases (50 per cent.) were in men, 16 (47 per cent.) in women; 39 per cent. occurred under the age of 30 (as compared with 18 per cent. of aortic aneurisms). Acute infectious diseases and lues seem to be the main etiological factors. The ductus arteriosus Botalli was frequently found open (17.5 per cent.), which would indicate that some disturbance during fetal life or soon after birth had been a predisposing factor. Frequently there is a certain degree of narrowing of the pulmonary artery (32 per cent.). In 8 cases (20 per cent.) there were also marked arteriosclerotic changes in the pulmonary artery.

Among 40 cases there were the following complications: pulmonary stenosis 2; relative pulmonary insufficiency 5; organic pulmonary insufficiency; other valvular lesions 3.

The subjective symptoms are not pathognomonic and are very similar to those of congenital heart disease; palpitation, dyspnœa and cardiac asthma, constriction of the chest, cough, often œdema of the lungs and blood-tinged expectoration, intense cyanosis, œdema, anasarca, ascites, hydrothorax. Death sometimes results suddenly from rupture, sometimes from intercurrent pericarditis and endocarditis, sometimes from diseases of the respiratory tract.

DIAGNOSIS.

According to Henschen, the diagnosis is justified when the following signs are all present simultaneously:

1. Intense cyanosis and other signs of stasis, constriction, and bloody expectoration, sometimes sternal pain.
2. Prominence of second and third left costal cartilages or second left interspace and well-defined dulness or X-ray shadow in this area.
3. Pulsation and well-defined thrill and murmur in second left interspace.
4. Loud superficial rasping systolic murmur.
5. Hypertrophy of the right heart.
6. Absence of dilatation or hypertrophy of left heart (apex dulness not outside the mammillary line).
7. Absence of other signs of aortic aneurism.

The X-ray shadow furnishes the most important aid in diagnosis. However, the correct diagnosis was made intra vitam only once or twice in his 40 cases.

ANEURISM OF THE ABDOMINAL AORTA.

Owing to its frequency (10–14 per cent. of aneurisms) and its surgical accessibility, aneurism of the abdominal aorta is of great importance. Owing to its exposed situation, trauma is a more frequent cause than in thoracic aneurism. As Sibson has shown, it is usually (133 out of 171 cases) situated just below the diaphragm and above the cœliac axis, in the place where it gives the greatest number of symptoms and is most inaccessible to operation.

The most important symptom of aneurism of the abdominal aorta is *abdominal pain*, epigastric or in the regions of kidney and gall-bladder, sometimes in the flanks, sides, and back. The pain is usually more marked on one side than the other, but may be bilateral. Until the appearance of a palpable tumor the condition may be readily mistaken for renal calculus, gastric ulcer, or other abdominal disease, or for psoas abscess. The pain may be so intense as to require morphine, even in large doses, though acetanilid, antipyrin, aspirin, etc., may be of use at first. Palpitation is also commonly felt in the aneurism.

All these symptoms are common in neurasthenic women who have vigorously pulsating abdominal aortas, especially associated with enteroptosis. It is probable that in this condition the peritoneal moorings of the aorta are rather loose. When the arterial pressure rises at systole, the angle curves of the abdominal aorta and common iliac arteries tend to straighten themselves and thus throw the aorta forward toward the abdominal wall, at the same time giving a painful tug upon the abdominal nerves as they emerge from the vertebral column. The looser the moorings of the aorta the greater its excursion and the greater the pull upon structures other than those which normally hold it. Arteriosclerosis of the abdominal vessels may also give rise to similar symptoms.

Mere pulsation of the aorta in the epigastrium and elsewhere, even when associated with quite intense pain, is therefore not necessarily a sign of abdominal aneurism. In doubtful cases *it is most important to outline the whole course of the aorta* by pressing the fingers of the two hands down on either side of the vessel so as to include the abdominal aorta between them. The expansile nature of the pulsation can be felt by pressing downward and inward. Any irregularity or bulging along its course may be felt readily in this way. For the *diagnosis of an aneurism it is necessary to outline a tumor with expansile pulsation* arising from the abdominal aorta, limited in extent above and below, and spherical or oval in shape. There is usually a well-marked thrill over an aneurism. The pulse-wave in the femorals is usually much retarded in aneurism (apex beat–femoral pulse interval = 0.24 sec.) but not in simple aortic pulsation. The early diagnosis may sometimes be made with the fluoroscope, care being taken to empty the bowels by a day or two free purgation and preliminary milk diet, and then to examine the abdomen with a "compression diaphragm" (Kompressionsblende) so as to push the other structures aside. Oblique illuminations and inflation of stomach and colon with air may be helpful.

As the aneurism grows it may press upon the renal arteries and veins and may cause *albuminuria*, cylindruria, hæmaturia, or even anuria

and death from this cause. It may press upon the intestines and cause intestinal paralysis, with death from obstruction, or may give rise to many symptoms from pressure. Erosion of the vertebrae and pressure on the cord or cauda equina may lead to paraplegia (flaccid) and may cause most intense pain.

Abdominal aneurisms may rupture the retroperitoneal tissue into the peritoneum, especially the lesser peritoneal sac into the stomach, intestines, or vena cava. They rupture externally in the epigastrium. The rupture is attended with excruciating pain and often collapse, but death may not ensue for some time thereafter, as the clotting of blood in a small space may prevent further outflow from the vessels. Thus, in the case cited below, the aneurism ruptured into the retroperitoneal tissue, compressing the renal vessels. The pain accompanying and following rupture was excruciating, probably owing to stretching of the solar plexus.

PROGNOSIS AND TREATMENT OF ANEURISM.

In spite of the fact that aneurisms occasionally cease to develop or even undergo spontaneous cure by thrombosis, this procedure is to be regarded as a rarity, and it is not, under any circumstances, to be expected. By far the greater number of aneurisms cause the death of the patient within from one to five years, though occasionally they remain stationary for twenty-five or thirty. It is, therefore, necessary to attempt to modify the course by treatment. As the intrathoracic aneurisms were not well known to the ancients, their therapy for aneurism was confined to ligature of the peripheral arteries.

Valsalva (1666–1723) recommended lessening the force of the heart-beat by absolute rest in the recumbent posture, "starvation diet." and frequent removal of small quantities of blood by venesection. The two former procedures were revived by Tufnell in 1874. Tufnell reported a number of cases, especially of aneurism of the abdominal aorta, cured by restriction of the daily intake to ten ounces of solids and ten ounces of liquids for several weeks.

Breakfast	Bread and butter	60 Gm. (℥ii)
	Milk	60 c.c. (℥ii)
Dinner	Meat	60–100 Gm. (℥ii–iii)
	Milk	75–125 c.c. (℥iii–iv)
Supper	Bread	60 Gm. (℥ii)
	Milk	60 c.c. (℥ii)

The patient is given no water, and is not allowed to rise from the horizontal position even for an instant. As a result of this the blood-pressure falls and the pulse-rate also. In his first case the pulse-rate fell from 104 to 69 per minute, equalling a diminution of 50,400 beats in twenty-four hours. The wall of the aneurism is spared just this amount of strain, the volume of blood diminishes, and the aneurismal sac may gradually contract down, facilitating clotting.

Tufnell's results (cure of two abdominal and one popliteal aneurism) are rather striking, but the treatment imposes the greatest hardship on the patient and few have the hardihood to give it an adequate trial. That the restriction of fluid to ten ounces daily may be harmful is suggested by

the fact that his first case developed uræmia at the end of the treatment and died from that about as soon as he would probably have died from the natural progress of the aneurism.

Alonzo Taylor has made very careful studies of the blood in three patients under Tufnell treatment, who were also receiving potassium iodide 1 Gm. (gr. xv) and calcium chloride 2 Gm. (gr. xxx) daily and who were being bled (250 c.c.) every eighteen days. He found no change in the concentration of the blood or in calcium in the blood, and only slight fall of red corpuscles. The coagulation time was unchanged in one patient, slightly shortened in another. The aneurisms became somewhat smaller, but no cures resulted. At the Johns Hopkins Hospital the method was tried very assiduously for many years, supplemented by subcutaneous gelatin injections as suggested by Lancereaux. Later calcium lactate has been used. There have been but few satisfactory results (Futcher), though Professor Osler stated that he had seen several cases of cure in his extensive experience.

Potassium iodide was used in aneurism by Bouillaud (1859) and Chuckerbutty (1862), and especially by Balfour, who found that it caused great relief from the pain, and claimed that the aneurism also diminished considerably in size. Subsequent experience demonstrates the correctness of the claim that aneurismal pains are often relieved by potassium iodide, but few, if any, cures of well-defined thoracic aneurisms can be obtained by its use. Its modus operandi is still obscure, but it may cause a retrogression of the luetic mesarteritis which is so often present. Gibson thinks that "we may admit it to be extremely probable that under the influence of iodide of potassium the nutrition of the walls of the sac, as well as of the whole of the arterial system, undergoes improvement."

Wiring.—The reason that an increased coagulability of the blood and a slowed circulation do not of themselves produce intrasaccular clotting is that the latter, like the vessels, is lined with endothelium and does not furnish fibrin ferment. As stated by Moore in 1864, "the first indispensable condition for the cure of a thoracic aneurism is to provide means of eliciting fibrin from the blood" (producing fibrin ferment *in situ*), and the "second . . . to extend the surface within it on which the fibrin may coagulate." In order to supplement these deficiencies, Moore suggested the introduction of fine wire into the aneurismal sac. Murchison submitted to him a case of aneurism of the ascending aorta which pointed on the surface of the chest. Moore slowly introduced twenty-six yards of fine iron wire through a fine needle. The pulse fell from 116 to 92, the pulsation of the tumor almost ceased, but the patient died in collapse two days later.

Previously to Moore, Guérard (1821), Pétrequin (1845), and Ciniselli (1847) induced clotting by passing weak electric currents between the tips of two needles introduced into the sac (galvanopuncture). Corrado combined the two methods by using the wire as one pole of the battery. As now performed, the wire (silver alloy) is attached to one pole of the battery. It is introduced through a needle which is covered with lacquer to prevent stimulation of the intercostal muscles, etc., and a weak current (10 milliamperes) is passed through it. This accelerates clotting and gives a firmer clot.

The details of technic, as well as an excellent report of the literature, are given by Hunner. Hunner cites 14 cases treated by wire alone (Moore's

method), with cure in 2 cases of abdominal aneurism (Morse and Langton); with the combined (wire plus electricity) method (Moore-Corradi), 23 cases—17 thoracic, with 3 cures (17.7 per cent.) (Dr. Rosenstirn informs the writer that his patient is still alive and well twenty years after the operation), and 6 abdominal aneurisms. Finney's case, who was apparently cured in 1900, died in 1903 of rupture of the aneurism after intense suffering for several years.

Unfortunately, the number of cases in which the Moore-Corradi method can succeed is a limited one. Its usefulness, as already pointed out by Moore, is limited to sacculated aneurisms of the aorta and to aneu-

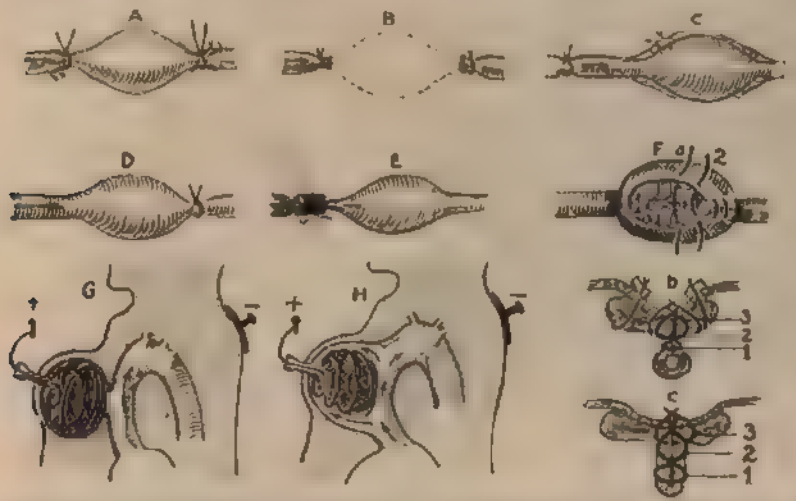

FIG. 315. Diagram showing the various methods for the operative treatment of aneurism. Arrows indicate direction of the blood stream. A, Ligature above and below the sac (Antyllus). B, Ligature above and below the sac, removal of the sac (Hoeter). C, Ligature above and at some distance from the sac (Hunter). V, V, represent vasa vasorum. D, Ligature below the sac (Brasdor, Wardrop). E, Metal band (Halsted). F, Obliteration of the sac (Matas). a, having the sutures in the sac. b, arterial tube left patent after tightening sutures. c, entire lumen obliterated by sutures. G, Successful wiring of a sacculated aneurism by the Moore-Corradi method, showing the electrodes [illegible] in place. H, Wiring of an aneurism with wide mouth, showing the action of eddy currents in penetrating between the clot and the aneurism wall. Compare with Fig. 316.

risms of arteries which have already been ligated below the aneurism (Brasdor's ligature, *e.g.*, wiring of an innominate aneurism after ligature of the right carotid and right subclavian arteries), so as to convert them into blind sacs.

The narrower the opening of the sac the greater the chance of permanent cure, for the clot then completely fills the sac and the blood passes by it without entering the old sac. However, if the sacculation has a wide mouth the condition is different. The whole sac may be filled by clot at the time of operation, but the irregularities about the edges of the clot give rise to eddy currents which gradually dissect the clot loose from the aneurismal wall, and leave as an end result an unaltered aneurism with an island of wire containing clot floating in the centre (Figs. 315, H, and 316). In an individual case the treatment will, of course, be more likely to succeed if preceded by a prolonged period of absolute rest, restricted diet and liquids,

and vigorous use of potassium iodide, so that the blood-pressure may be as low, the size of the sac as small, and its neck as narrow as possible. The same treatment should follow the operation to give a chance for the most compact adhesion of the clot to the aneurismal wall.

In fusiform aneurisms the wiring method is worse than useless, since it merely gives rise to a clot in mid-blood stream from which emboli are readily dislodged.

Compression. — Aneurisms of the peripheral arteries, and especially of the abdominal aorta, are sometimes cured by compressing that vessel above the aneurism. This was done successfully by Murray in 1864, who obliterated the aneurism and the femoral pulse by means of a tourniquet wound around the body above the tumor. A number of similar successful cases have been reported since Murray's, especially when the aorta is compressed with the fingers. The operator cannot continue digital compression longer than five or ten minutes at a time, so that it is often the custom to obtain the assistance of a whole class of medical students working in relays. In this way Shepherd and others have been able to keep the aorta occluded for twenty-four hours, and have brought about recovery.

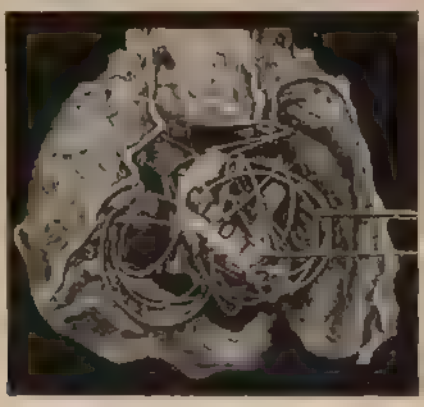

FIG. 318.—Specimen of wired abdominal aneurism showing an olive of clot within the coils of wire surrounded by a free blood-channel.

On the other hand, the prolonged pressure may bring about necrosis of the abdominal wall, intestine and pancreas, or secondary peritonitis, and intestinal obstruction may result (Bryant, Lunn and Benham, Moxon and Durham). The method is therefore still a daring one, and is probably more severe and less certain than Halsted's metal band method. Moreover, Sibson found 133 out of 177 abdominal aneurisms (75 per cent.) above the level of the coeliac axis where they could not be reached by pressure.

Ligature and Partial Occlusion.—Double Ligature.—The oldest method of treating aneurisms of the peripheral arteries is to ligate them above and below the sac (Antyllus) (Fig. 315, A), after opening the latter to remove the blood. A more modern modification of this method is that of Hueter, who dissected out the entire sac after ligating, thus removing a large mass of tissue which would otherwise become gangrenous.

Proximal Ligature. Ambroise Paré (sixteenth century) departed from the procedure of Antyllus by merely ligating the artery close above the aneurism (proximal ligature—close to the aneurism). This cut off the blood supply to the walls of the latter and to its vicinity, inducing necrosis and suppuration, so that Anel (1710), Desault (1785), and John Hunter (1785) were led to adopt the proximal ligature at a considerable distance above the aneurism (ligature of brachial for aneurism of the radial artery;

ligation of femoral below the adductors for popliteal aneurism; ligation of femoral above the adductors—in Scarpa's triangle—for popliteal aneurism).

Distal Ligature.—In the eighteenth century Brasdor and later Wardrop practised ligation of the artery below the aneurism (distal ligature) in cases like aneurism of the innominate in which the proximal ligature was impossible. As a result of the procedure, a fusiform aneurism of the innominate becomes practically a sacculated or flask-shaped aneurism of the aorta, the innominate artery being converted into a blind sac with narrowed neck, and coagulation is thus facilitated. This operation is still the one most commonly performed for aneurisms of the innominate, carotid, and first part of the subclavian artery. Sheen has collected statistics of 36 cases of innominate and subclavian aneurism, 22 before 1880 with 1 recovery, 14 after 1880 with 8 recoveries, 7 after 1890 with 5 recoveries and 5 cures. To this list might be added 2 cases of Halsted and 1 of Finney with recovery and cure. The deaths before 1880 were usually due to sepsis and hemorrhage. In operating upon the innominate artery it is most important that both the carotid and subclavian arteries should be ligated, for if one of these arteries be left open (as in the case of J. B.) the pressure in the sac is increased without stopping the blood-flow through it, and the growth of the aneurism is actually favored.

Moore in his first paper suggested the combination of this form of ligation with wiring for aneurisms of the innominate. This double procedure has not attracted much attention, as in the absence of sepsis the simple ligature is often satisfactory, but it is no doubt applicable in a certain number of cases where ligation is not quite adequate.

The chief objection to the simple ligation of arteries lies in the fact that the permanent results are often unsatisfactory, for either the ligature may be so tight as to produce necrosis of the tissues under it and thus bring on rupture of the artery, or, as Halsted has shown, the arterial lumen may be re-established in spite of the ligature. In many of Halsted's experiments upon ligating the larger arteries, the lumen of the artery gradually dilated above and below the ligature, so that the latter was left surrounded by a thin membrane or septum of scar tissue. This septum then perforated in one or two places.

Occlusion with Metal Bands. To obviate this and for other reasons Halsted has devised a very ingenious procedure, which consists in the occlusion of the vessel by surrounding it with wide metal bands. These metal bands take the place of the ligature; but when properly applied, do not occlude the vasa vasorum, and hence permit the proper nourishment of the arterial wall. Their effect is more certain than that of ligatures, since they do not allow the lumen to be re-established. The chief advantage, however, lies in the ability of the operator to obtain a partial occlusion of the vessel sufficient to reduce pulsation in the aneurism to any desired degree, without obliterating the circulation below before a collateral circulation has been established. This renders it the operation *par excellence* in abdominal aneurisms and aneurisms of the iliac and femoral arteries, in which the other procedures are likely to be dangerous.

The bands are made of No. 33 sheet aluminum of a width varying from 5 to 15 millimetres according to diameter of the artery which is to be occluded. The strip is cut a little

longer than the circumference of the artery (as shown by a tape passed around the artery). All the sharp edges must be carefully filed off until they are smooth and round, lest they cut into the walls of the artery. The strip is then inserted into a specially devised holder (Fig. 315, E), where it is held in a slot; from this it may be extruded by pressing upon the rammer above, and as it is extruded below it is curled by the curve at the foot of the slot. The faster the strip is extruded the more tightly it is curled. The curved foot of the holder is placed beneath the artery, which is held just tightly enough against the instep of this foot to almost occlude the lumen. The strip is extruded by pushing the rammer just fast enough to give the desired curl. Tension on the artery is then relaxed. The pulse can be felt in the artery below the band, accompanied by a well-defined thrill. The band is then tightened by rolling it gently under the fingers of one hand while palpating the artery below it with the other. The degree to which the band is tightened depends upon the artery affected. In the case of the abdominal or descending thoracic aorta it should be pulled until the thrill has greatly diminished but not disappeared, in the larger branches of the aorta the pulse may be made to disappear absolutely. In a few minutes a regurgitant pulse may mark the appearance of a collateral circulation. When this operation is performed successfully, the artery becomes gradually occluded at the point of constriction and a rich collateral circulation formed, so good in fact that in one case in which Professor Halsted had occluded the descending thoracic aorta Erlanger found the blood-pressure in the femoral (eight months afterward) only thirty millimetres below that in the brachial. For practical purposes this exactly duplicates the conditions in the adult type of stenosis of the isthmus of the aorta (see page 452), except that the anastomoses take place later and hence are not quite as extensive.

Professor Halsted has now operated upon a number of cases with very promising results, and the operation gives promise that in the hands of a surgeon who has practised the technic, it may completely supersede the methods of ligature and compression.

Arteriorrhaphy (Matas Operation).—R. Matas in 1905 introduced an entirely new technic in treating the aneurism of peripheral arteries exactly in accordance with the principles of treating inguinal hernia—by obliteration of the sac. The operation is performed bloodlessly.

The limb is elevated, an Esmarch rubber bandage put on, or bleeding from the main artery prevented by compression with a traction loop, adjustable clamps (Crile's), padded forceps or digital compression. A free incision parallel to the long axis of the sac is then made down to the sac to expose its whole length. Any important nerves or veins should be dissected away from it. The sac is then freely opened and emptied. It is then ready for closure. In most cases it will be decided to obliterate the sac completely, but in some cases of fusiform aneurism it may be preferable to leave a lumen the size of the original artery.

When the sac is to be completely obliterated, the lining of the sac is thoroughly scrubbed over its whole extent with sterile gauze soaked with salt solution to remove the endothelial layer of the intima, and thus accelerate union. The sutures (chromicized gut) are then applied very much like Lembert's intestinal sutures. The most important point is to approximate carefully intima to intima. The sutures are laid in three layers in such a way that the cross-section of the sac after suture is made to form a Y, the first and deepest layer of sutures shutting off the sac from the artery at the Y, and the third layer obliterating the cavities in each arm of the Y (Fig. 315, F).

In suturing a fusiform aneurism the lumen of the artery is preserved by placing the first layer of sutures over a rubber tube which is inserted into the artery. After the new lumen is thus provided for, the rest of the sac is scrubbed and the sutures laid in the usual way. Care must be taken to preserve the blood supply and nutrition of the sac, and all portions of it which have been dissected away from their vascular surroundings should be excised.

In 1908 Matas reported the results of 86 such operations, including aneurisms of the femoral, the iliofemoral, tibial, gluteal, external carotid,

axillary, brachial, and subclavian arteries, as well as the abdominal aorta (the latter both fatal): 78 recoveries; 8 deaths; 4 cases of gangrene; 4 relapses, all in operations where the lumen was restored. In view of the fact that these 86 operations were performed by fifty-two different operators the excellent results obtained are a striking argument in favor of the feasibility of the operation.

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PART IV.

I.

PAROXYSMAL TACHYCARDIA.

Cotton in 1867 described a peculiar condition in which attacks of extreme tachycardia were present, leaving the heart quite normal in the interim. Similar cases were reported by Benson, Nothnagel, Proebsting, Priesendorfer, Pribram, and Bristowe, who considered them to be due to a sort of vagus neurosis. Bouveret, however, regarded the condition as a distinct clinical entity, of which he was able in 1889 to collect over twenty cases from the literature, and which he designated as "essential (or idiopathic) paroxysmal tachycardia (tachycardie paroxystique essentielle)". According to Bouveret, this condition is characterized by attacks in which the pulse suddenly attains a rapidity (200 to 300 per minute) which is never seen in any other condition, even in the gravest heart failures. These attacks last from several minutes to several days or even weeks, and subside as suddenly as they come. They sometimes recur for years and often for decades without seriously interfering with life and general health of the patient; or, on the other hand, an attack sometimes ends in death.

Bouveret's clinical description was so complete that, though many cases were subsequently reported, little that was essential was added until Aug. Hoffmann in 1900 called attention to the fact that the paroxysms of tachycardia began and ceased with extreme suddenness, and showed by excellent tracings that the complete change of rate often occurred within the period of a single cardiac cycle. Moreover, he showed that this change of rate was an exact doubling, trebling, or quadrupling of the previous rate, and ended by halving, quartering or dropping to one-third. For example, the normal pulse-rate being 70, the rate during an attack might be 140, 210, 280 per minute, and *vice versa*. Hoffmann regarded this sudden complete change of rate as characteristic of the essential or idiopathic paroxysmal tachycardia, in contrast to the simple tachycardia of exercise, excitement, or convalescence, in which the change of rate is due to loss of vagus tone and comes on by a gradual increase of rate during a period of from one to several minutes. Such a tachycardia rarely exceeds 120 to 140 per minute. Even though it may give rise to sharp attacks coming on more or less suddenly and accompanied by palpitation, it is not to be regarded as idiopathic (essential) paroxysmal tachycardia, but will be considered under the simple nervous affections of the heart (Chapter III).

As will be seen, one cannot lay too much stress upon the importance of distinguishing between "paroxysms of tachycardia" and "idiopathic paroxysmal tachycardia." Only those cases should be considered in which the mode of onset and cessation of the attack is carefully given, if possible with venous pulse tracings. The mechanism involved in the attacks should also be noted. It is only in this way, and not by indiscriminate analyses of cases in which the heart occasionally becomes rapid, that an accurate knowledge of the condition can be acquired. The accurate knowledge of paroxysmal tachycardia, therefore, dates from Hoffmann's analysis of pulse tracings.

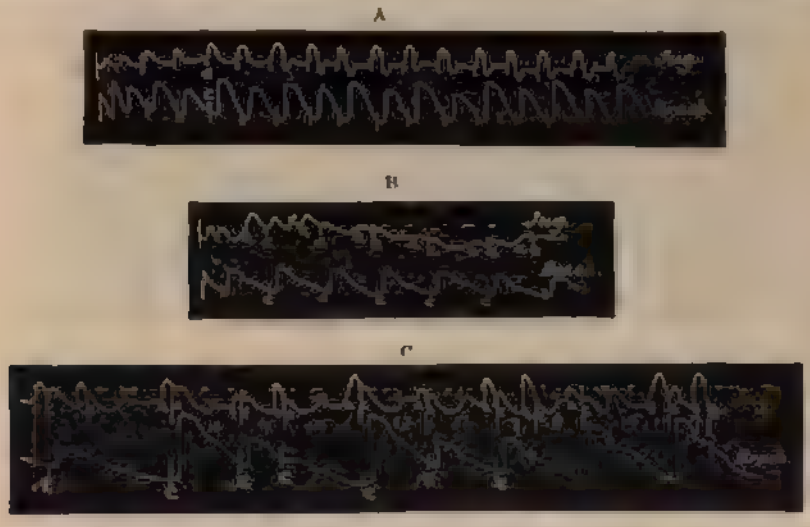

FIG. 317.—Venous pulse in a case of paroxysmal tachycardia (G. D. R.). (Kindness of the Johns Hopkins Hospital Bulletin.) A. During the attack (pulse-rate 144 per minute). Ventricular type of venous pulse: no *a* wave discernible; *c* carotid wave. Time of the carotid wave. B. Tracing taken five minutes later, just after cessation of the attack. Pulse-rate 90. Venous pulse of the normal auricular type; conduction time, *a-c* interval, normal. C. Tracing from the same case (taken during a period of irregularity a few days later, showing extrasystoles with shortened conduction time. The intervals are measured in millimetres upon a uniformly running drum.

Still more accurate knowledge came with the analysis of venous tracings as well, and of tracings obtained at the moments when the attacks began and ceased.

Types of Paroxysmal Tachycardia.—By this means several types of venous tracings have been recorded:

1. Attacks of tachycardia in which the auricular type of venous pulse remains, and in which, at the cessation of the attack, the auricles continue for a while at least at their old rhythm, the rate of the ventricles falling to half or less by the onset of a partial auriculo(atrio)ventricular block and a 2 : 1 rhythm (cases reported by Hoffmann, Gerhardt, Rihl, and Schmoll). There may be periods of irregularity between, especially just before and just after, attacks due to the occurrence of partial block for occasional beats. The partial block even in these cases does not persist indefinitely, but the rate of the auricles finally also becomes slow, and a 1 : 1 rhythm at the slow (normal) rate is resumed.

2. Attacks in which the venous pulse is of the ventricular type (see page 57) with no wave due to auricular contraction, and which subside suddenly with approximate halving or quartering of the rate without signs of auriculoatrioventricular block, the venous pulse between attacks showing only a single auricular wave for each ventricular contraction. Between attacks there may be an irregularity due to the presence of extrasystoles with shortened conduction time. Cases of this type have been studied by Mackenzie, Hirschfelder, Hay, and others.

A careful analysis of the pulse-rate during and between attacks shows that the rate is by no means always a definite multiple, but varies within

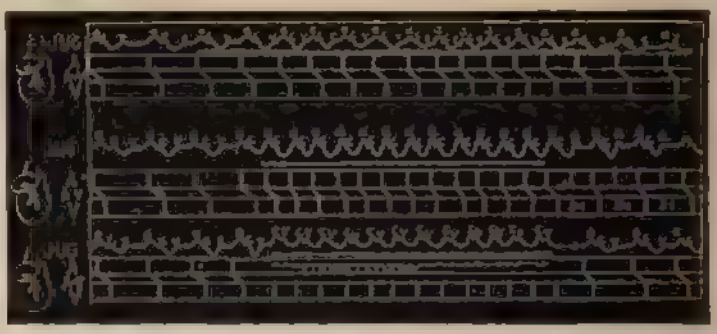

FIG. 318.—Diagram showing the various types of tachycardia. I Simple non-paroxysmal tachycardia, showing the gradual increase and gradual decrease in rate. *J V C* venous pulse. A, auricular impulses. V ventricular impulses. A-V, auriculoventricular conduction. II Paroxysmal tachycardia with persistent auricular contraction and ending in auriculoventricular heart-block. 2 : 1 rhythm. III. Paroxysmal tachycardia with auricular fibrillation and ventricular type of venous pulse.

considerable limits (as for example, from 1.7 to 2.1 : 1; 88 to 140 and *vice versa* 70 to 160) even when tracings are obtained from the instant of onset or of cessation of an attack.

OCCURRENCE AND ASSOCIATED LESIONS.

Paroxysmal tachycardia is equally common in both sexes (Bouveret, Hoffmann). It occurs at all ages, frequently beginning in early childhood and persisting for decades. On the other hand, it frequently occurs in old persons, as in G. R. who was 72 years of age. It is not usually associated with valvular or organic disease of the heart, though the occasional attacks of sudden tachycardias often seen in aortic insufficiency may belong to this group.

Sometimes, as in the cases reported by Romberg, there is associated coronary sclerosis—a group which seems to be particularly common. This seems to correspond to the experimental observation that shortly before death the auricles of the exposed dog's heart sometimes pass into fibrillary contractions for a short period upon the slightest mechanical irritation, as well as to the experiments of T. Lewis. The writer has encountered a number of cases associated with mitral stenosis.

The autopsy findings of Mackenzie and Keith, patches of fibrous myocarditis in the vicinity of the His bundle and coronary sinus, are of great interest, but await further observations before they can be accepted as the

pathogenetic lesion. On the other hand, tumors, patches of fibrosis, and arteriosclerosis in the vicinity of the vagus nucleus in the medulla, adhesions along the course of the vagus (Reinhold, Hoffmann, Pitres, Oppenheimer, Schlesinger, Pal), multiple sclerosis (Müller), early tabes (Hirschberg) are sometimes found. However, S. Hyman, in Sir Victor Horsley's laboratory, has produced permanent lesions of the vagal nuclei in a series of dogs and monkeys without ever giving rise to paroxysms of tachycardia. Nevertheless, it is conceivable that continued reflexes from irritation of nerves through pressure of tumors from hernias, intestinal parasites, etc., may increase the irritability of the heart muscle just as they often increase that of the cerebral cortex. Indeed, paroxysmal tachycardia bears certain superficial resemblances to a condition of "epilepsy of the heart," and is occasionally associated with idiopathic epilepsy (Nothnagel, Schlesinger). In a number of cases collected by Hoffmann disturbances of the digestive and pelvic organs were found, in some cases floating kidney. In some

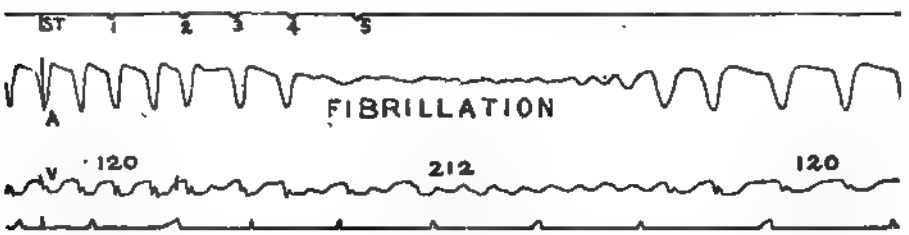

FIG. 319.—Experimental paroxysm of tachycardia produced by faradization of the dog's auricle. (Kindness of the Journal of the American Medical Association.) *ST* 1, 2, 3, 4, 5, faradic stimulation of the right auricular appendix; *A*, auricular systoles; *V*, ventricular systoles. Time in seconds.

cases the attacks date from an acute cardiac overstrain; in others from an attack of rheumatism, with or without other cardiac complications. However, the writer can confirm the statement of Hoffmann, that in many cases neither the underlying condition nor the factors bringing on the attack can be discovered.

Onset.—The attacks themselves often occur at the moment of awaking from sleep, after or during defecation, during conditions of fatigue, and are sometimes brought on by nervous excitement, as was the case in the second attack of one of our patients. Mere percussion of the precordium has been known to bring on an attack (Bouveret). Change in the position of the body from the horizontal to the vertical may bring on an attack (orthostatic paroxysmal tachycardia).

NATURE OF FUNCTIONAL DISTURBANCES.

The nature of the functional disturbance is still obscure, though several theories have been advanced. Hoffmann, Lommel, Gerhardt, and Mackenzie at first believed that it consisted in the interpolation of an extrasystole between each two regular systoles, calling attention to the fact that the arterial pulse often showed an alternation of large and small beats; but Bayliss and Starling, Hirschfelder, and others have been able to show that this indicates that the rate is a little

too fast for the optimum contractions of the ventricles rather than that the small systoles are of abnormal origin.

Hoffmann (1904) suggested that the sudden change of rate (to approximate multiples or fractions of that pre-existing) represented the coming on or the passing off of a block between the site at which the cardiac impulses arise (remains of embryological sinus, the area bounded by the venæ cavæ, coronary sinus, and septum auriculorum and in front by the Eustachian valve) and the auricular muscle tissue—a true sino-auricular block. Just as in the cases of atrioventricular block above mentioned, the attacks would thus correspond to the periods when the block has passed off, the return to normal pulse corresponding to the onset of block. However alluring this theory may be, it must be admitted that there is at present little evidence to support it.

More plausible is the theory of Mackenzie (1903-04), that these attacks are brought about by a condition in which the (Purkinje) cells of the His auriculo- (atrio-) ventricular bundle initiate the rhythm of the beat instead of the sinus. This theory is founded not only upon the above-mentioned venous tracings, but also upon the statement of Gaskell that, if one "touch the auriculoventricular ring of muscle (in the frog) with the slightest stimulus, immediately a series of rhythmical contractions occurs," while touching the auricular and ventricular muscle causes only a single contraction in each case. In mammals, however, the evidence upon this point is very flimsy, for it has been impossible to stimulate the fibres of the atrioventricular bundle alone without including fibres of the auricles and ventricles. Lohmann, it is true, stimulated the region of the auriculoventricular bundle (including the auricular and ventricular muscle) and obtained simultaneous contractions of the auricles and ventricles, which outlasted the period of stimulation. Erlanger has obtained somewhat similar results, but does not regard them as conclusive. Hering and Rihl obtained extrasystoles with shortened conduction time and *assumed* that they arose in the bundle of His. Mackenzie and Keith, however, claim to have found deposits of cells whose cicatrization "irritates the bundle and renders it more excitable than the sinus. The contraction of the heart then originates from this more irritable part. Somewhat analogous changes follow in cardiosclerosis and in degeneration of the coronary arteries."

Hirschfelder, however, has been able to duplicate exactly the findings of Lohmann and Hering by faradic stimulation of the exposed dog's auricle, not in the vicinity of the auriculoventricular bundle, but far out upon the auricular appendix. Under these conditions he obtained the following results. Very weak faradic stimuli caused the occurrence of ordinary auricular extrasystoles with normal conduction time, slightly stronger stimuli caused the auricles suddenly to assume a rapid regular rhythm approximately double the previous rhythm (the ratio varying from 1.7 to 2.1 to 1). The ventricles usually followed perfectly. Conduction time was prolonged. Long continuance or repetition of this stimulation or increase in the faradic stimulus increased the irritability of the auricular muscle (perhaps also that of the Purkinje fibres). The first effect was shortening of the conduction time. With still more increased or more frequently repeated stimuli the auricles went into fibrillary contractions (delirium) upon slight stimulation, the fibrillation, at first lasting only during the period of stimulation,

later or with stronger stimuli, outlasting the period of stimulus. When the stimulus and the abnormal auricular contractions were only instantaneous, extrasystoles with shortened conduction time were present (auriculo(atrio)ventricular extrasystoles), just as in the writer's case of paroxysmal tachycardia. When the fibrillation was prolonged, a long period of ventricular tachycardia accompanied it, sometimes lasting for many minutes, and ending by a sudden return to the original rhythm with normal auricular rate, force, and normal conduction time. In the less irritable hearts these attacks may be brought to a standstill by stimulation of the vagus, but as irritability increases the tachycardia returns. It is found that the whole heart may be stopped by vagus stimulation, but the tachycardia is resumed as soon as vagus stimulation ceases! Moreover, Cushny and Edmunds who have investigated paroxysmal irregularities with tachycardia in man and animals have found that, just as in the cases of paroxysmal tachycardia, the venous pulse assumes the ventricular type during fibrillation of the auricles. This condition represents the nearest approximation to paroxysmal tachycardia which has been produced experimentally, and has led the writer to the hypothesis that "true (idiopathic) paroxysmal tachycardia is usually caused by some one of a number of conditions which bring about a state of increased irritability of the heart muscle especially of the auricles, which may pass into a state of fibrillation." It is possible that cells of the atrioventricular bundle may either primarily or secondarily give rise to the impulse.

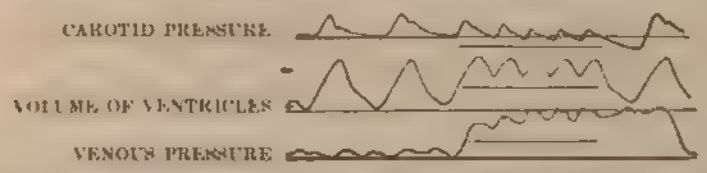

FIG. 320.—Diagram showing the effect of a paroxysm of tachycardia upon the circulation. The underlined portion indicates the paroxysm.

Whether the stimulus originates in the Purkinje fibres or elsewhere seems for the present to have little practical importance. More important is the fact that Reid Hunt, Cushny and Edmunds, Garrey and Hewlett, and the writer have observed that such paroxysms may occur not only spontaneously or from direct stimulation of the heart muscle, but also upon stimulating the cardiac nerves, either accelerator or vago-sympathetic, in hearts whose irritability is already abnormally high. Hence it is natural that when the cardiac irritability is high, small reflex stimuli bring about an attack.

Stimulation of Cardiac Nerves. That something more than mere neurogenic influences is essential was shown by Gerhardt and Hirschfelder. These observers paralyzed the vagi of such patients with atropine and yet produced no attack. Hirschfelder found that slight stimulation of the accelerators, by exercising his patient to the point of giddiness twenty-four hours after an attack and while his vagi were paralyzed with atropine, caused a slight gradual increase of pulse-rate, but nothing resembling an attack of paroxysmal tachycardia. The condition of extreme irritability of the cardiac muscle had evidently passed off.

Paroxysmal Tachycardia from Coronary Ischæmia.—Quite recently T. Lewis (Paroxysmal Tachycardia. Heart, Lond., 1909, i, 42) has succeeded in producing paroxysms of tachycardia in cats and dogs by ligating one of the coronary arteries, especially the right. These attacks come on even after section of both vagosympathetic nerves and hence were not neurogenic in origin. He has been able to obtain four types of abnormal cardiac cycles

during such paroxysms: 1. Approximate doubling of the rates of both auricles and ventricles with normal or slightly diminished conductivity. 2. Approximate doubling of these rates of these chambers, but the auricles and ventricles contract simultaneously (nodal rhythm?). 3. Fibrillation of the auricles with approximate doubling of rate in the ventricles. 4. Approximate doubling of rate in both auricles and ventricles, but the ventricles contract before the auricles (ventricular tachycardia). The writer has also obtained all of these four types during faradization of the auricles, so that it would appear that they may all be traced to a common cause, most probably over-excitability of the heart muscle. Moreover, Rothberger and Winterberg have obtained electrocardiograms with small irregular waves, suggestive of auricular fibrillation, from a patient with paroxysmal tachycardia; and Professor Barker, Dr. Bond, and the writer have recently obtained a similar tracing from a patient between paroxysms, though electrocardiograms from another patient in the midst of a severe paroxysm showed apparently normal contractions of the auricles. Rothberger and Winterberg have obtained exactly similar electrocardiograms from animals with exposed hearts in which they produced a state of auricular fibrillation by faradization of the auricles.

This coronary form of paroxysmal tachycardia is exactly similar to the cases of Romberg and Barker (page 283), but it is by no means certain that this factor is the causal one in all cases of paroxysmal tachycardia.

EFFECTS ON CIRCULATION.

The effect which these paroxysms of tachycardia exert upon the circulation is primarily due to the deficient filling of ventricles during the short diastoles. As Yandell Henderson has shown, the ventricles fill to their normal extent only when the pulse-rate is moderately slow. When the pulse becomes rapid, the ventricles do not have time to fill, and, since the period of systole is never much less than 0.2 second, it is evident that when the heart-rate is much above 200 the period during which filling can take place is very short and little blood can enter the ventricles. The volume of the heart remains small. As a result of this condition, blood stagnates in auricles and veins, venous pressure rises (to 30 cm. H_2O in one case examined by Eyster and Hooker), and with it there come engorgement of the liver and œdema of the extremities. Stasis also occurs in the pulmonary veins, ushering in œdema and dyspnœa, and sometimes these symptoms of broken pulmonary compensation dominate the scene (see page 139).

On the other hand, the arterial pressure falls, because, as the filling of the ventricle is small, the amount which is driven out into the arteries is diminished correspondingly. This fall in blood-pressure is usually accompanied by pallor and often by symptoms of cerebral anæmia, exactly as occurs in hemorrhage, surgical shock, or other conditions in which the amount of blood in the arteries is diminished. Other organs also suffer from anæmia, and finally also the heart itself, which may give signs of weakening, first evinced by lowered tonus and dilatation. It is evident that hearts whose coronary arteries are sclerotic would suffer more readily than those with normal blood supply.

PHYSIOLOGICAL SUMMARY.

The pathological physiology of paroxysmal tachycardia may therefore be summed up as follows.

Underlying causes: Increased irritability of cardiac muscle.

Predisposing factors for an attack. Slight reflex stimulations of cardiac nerves.

Condition during attack: "Doubling" or multiplication of pulse-rate, with or without auricular fibrillation.

Mechanical effects on the circulation, to which these symptoms are referable:

1. Systemic stasis, high venous pressure.
2. Pulmonary stasis, high pressure in pulmonary veins.
3. Anæmia of brain, kidneys, and heart, from low arterial pressure.

SYMPTOMS.

Although attacks of tachycardia sometimes run their course without the patient's knowledge, it is more common for them to be accompanied by symptoms. These symptoms may be grouped as follows:

1. Symptoms of cardiac excitability.
2. Those due to engorgement of systemic veins (failure of right ventricle).
3. Those due to engorgement of pulmonary veins (failure of left ventricle).
4. Those due to cerebral anæmia.

1. Palpitation, a feeling of discomfort or oppression in the precordium, and weakness are the most common symptoms. This is often worse just at the end of the attack, and may, as in Hay's case, resemble the symptoms of angina pectoris. In this case there was also hyperæsthesia of the precordium and neck. The latter may be due to engorgement of the cervical veins, as in angina pectoris it may be referred from the heart (Mackenzie).

2. Besides the above-mentioned feeling of fulness in the neck, the patient often has a similar feeling in the abdomen from distention of the liver, and swelling of the feet commonly appears before the end of the attack.

3. Dyspnoea is frequent. It is striking that this may occur without any change in the rate of respiration, even in cases with severe myocardial changes (Romberg). No doubt this is associated with engorgement and high pressure in the pulmonary veins. It is often accompanied by cough and the expectoration of mucus, sometimes containing large endothelial cells with blood pigment (Herzfehlerzellen). Occasionally there is actual hæmoptysis (in three of Bouveret's eleven cases) during the attack. Actual pulmonary œdema may indeed set in, as in Přibram's case,—a young woman otherwise healthy, whose attacks were so severe that "the pulse became barely palpable. The patient fell into a state of collapse, and finally œdema appeared in the lower part of the lungs. At the moment when death seemed imminent, when collapse was at its height, she gave a cry of anguish; it seemed to her as though something were taken out of her neck, and the scene suddenly changed. The pulse fell to 76, became large and full, and the collapse disappeared."

On the other hand, this sudden change does not always occur, and death sometimes supervenes during the attack.

The venous stasis also leads to albuminuria, though in the milder attacks the urine may be increased and of low specific gravity.

4. The fall in arterial pressure usually brings about *symptoms of cerebral anaemia*; weakness, vertigo, and even extreme nervousness is the rule during the attacks, accompanied by restlessness, loss of appetite, and inability to sleep. Even syncope may occur. In a gentleman whom the writer examined some years ago these syncopal attacks had led several prominent physicians to diagnose Adams-Stokes syndrome, when, as was shown by the examination and subsequent observation, the cerebral anaemia resulted not from bradycardia but from tachycardia. Fortunately, these attacks have a tendency to become milder. Dr. Lyon writes, three years after the first examination, that this patient "is now able to play cards, go fishing, and do almost anything in a quiet way during attacks."

PHYSICAL SIGNS.

Physical signs are absent between attacks of paroxysmal tachycardia. During the attacks the face is usually pale, the expression anxious, the pupils are equal, the veins of the neck are seen to be engorged and often to show a *positive "single" pulsation* accompanying each systole (sometimes due to transitory tricuspid insufficiency), perhaps due to the feebleness of the auricular contractions. The tumultuous heart action is often seen in a precordial heaving and well-marked apex beat. The area of cardiac dulness is rarely increased except toward the end of the attack. It is *usually unchanged or decreased in size*, corresponding to the diminished filling of the ventricles. This *diminution* in the size of the heart during an attack has been seen with the fluoroscope by Hoffman, Dietlen, and others, and it can be demonstrated in the experimental paroxysms. Towards the end of severe attacks dilatation sets in from cardiac weakness.

The heart sounds may be unchanged, but usually become short and somewhat muffled. There is often embryocardia. It is very common to hear a soft systolic over the right ventricle and apex, perhaps due to a mitral or tricuspid insufficiency of the papillary type.

Sometimes the cardiac rhythm is irregular, owing to inability of the ventricles to follow all the impulses from the sinus and auricles or to the presence of extrasystoles. The liver often is felt to be enlarged, and often shows a systolic pulsation during attacks (tricuspid insufficiency), but ascites rarely occurs. Œdema of the ankles and feet is very frequent.

CASE OF PAROXYSMAL TACHYCARDIA.

G. D. R., a hotel-keeper aged 72, was admitted to the Johns Hopkins Hospital on Feb. 22, 1906, complaining of palpitation of the heart. The family history and personal history were negative. The patient had always been a robust man, had had no infectious diseases and no other cardiac manifestations.

The *first attack of palpitation* and tachycardia came on suddenly after retiring one evening *twenty years before admission*. It caused him great *fear*, but no pain. The attack lasted six hours and *left him weak but otherwise well*. Attacks similar in character recurred once a month until the winter of 1905-1906, when they became more severe and began to occur once or twice a week. During the attacks he passed large amounts of urine. He never noticed palpitation between attacks of tachycardia.

The patient was a large well-nourished man of good color. His pupils were equal and reacted well to light and during accommodation. The thorax was rather barrel-shaped, the percussion note was hyperresonant, and the breath-sounds were clear, though distant. The cardiac impulse was neither seen nor felt, but the apex, as made out by percussion and auscultation, was situated in the fifth left interspace 11 cm. from the midline. The cardiac dulness extended up to the upper border of the third rib, but could not be made out to the right of the sternum. The sounds were distant, but clear. The pulse-rate between attacks was 64 per minute. It was usually regular in force and rhythm, of good volume and rather high tension; the blood-pressure ranged from 165 to 190, the minimum from 100 to 115.

The abdomen was large and flabby, with considerable panniculus. Liver and spleen were not palpable. The examination was otherwise negative. The venous pulse between attacks was usually normal.

On Feb. 23 and March 1 and 15 the patient had attacks of tachycardia, in which his pulse-rate rose suddenly from 80 to 88 per minute to a height of 144 to 160 per minute. The attack of March 15 began just after returning from the closet where he had passed a soft fluid stool. Tracings made from the patient during this attack showed what is probably a ventricular type of venous pulse during the attack. When the latter ceased, however, the pulse resumed the normal auricular type. There was no sign of auriculoventricular block. Excitement incident to being shown at the clinic precipitated a second attack on March 15, which was not relieved by the application of an ice-bag, yawning, deep breathing, pressure on the vagus, in front of the sternocleidomastoid nor by administration of spiritus ætheris nitrosi, amyl nitrite, or digitalin. The attack ceased spontaneously within an instant, at 3.10 p.m.

On March 21 his pulse was irregular, due to the presence of numerous extrasystoles with shortened conduction time (auriculoventricular?). These subsided, however, leaving his pulse regular. On March 24 his pulse remained at 76 in spite of the administration of 2 mg. atropine. Even rapid walking while he was under the influence of the atropine did not bring on an attack, nor did the administration of amyl nitrite on March 24.

A very well-defined case of tricuspid insufficiency resulting from the cardiac overstrain of a prolonged paroxysm of tachycardia is exemplified by the following patient seen in consultation with Professor Barker.

Case of Long-standing Paroxysmal Tachycardia

W. W. C., clerk in the U. S. Patent Office, aged 29, had always been healthy except for a very severe attack of gonorrhœa six years before admission. He had no cardiac disturbance until seven years ago (one year before the attack of gonorrhœa), when he had symptoms of slight cardiac weakness which was said to be valvular (?) but these soon disappeared under treatment so that he was able to dance and take all kinds of exercise without symptoms. Two years before admission he awoke one morning after an emission, with severe palpitation and a very rapid weak pulse. He was kept quiet, an ice-bag put to his chest, and he was given strychnine 1.5 mg. (1/40 gr.), also tincture of strophanthus. His pulse-rate dropped to 72. Three weeks later he had another emission and another attack, and since then had a large number. The attacks often come on after emissions which leave him feeling very much depressed. They subside very suddenly and the pulse returns to normal at a bound, remaining between 70 and 100 per minute between attacks. In the present attack, however, the pulse-rate has been rapid continuously for over a year (since April, 1908), and this has been accompanied by palpitation and great weakness, occasionally by nausea and vomiting. It has not been relieved by strophanthin, digitalis, strychnine, nitroglycerin, belladonna, or potassium bromide.

The patient is a pale nervous-looking young man. The pupils are rather wide, but there are none of the ocular signs of Basedow's disease. The thyroid is not enlarged. There is no glandular enlargement. His chest is long and rather flat, but shows nothing of importance.

The heart is much enlarged, the apex being located in the sixth left interspace 10.5 cm from the midline. It is very movable within the chest-site altering 5 cm as the patient turns from side to side. There is a heaving impulse over the precordium, with systolic retraction of the interspaces over the right ventricle and marked systolic retraction in the epigastrium. Dulness extends above to the second interspace at the left sternal margin, and to the right reaches 5 cm from the midline. Longitudinal diameter, 20 cm. The area of flatness extends from the apex to the level of the fourth rib and just beyond the sternal margin in the fifth right interspace. The heart sounds are heard at the apex, the first being accompanied and followed by a slight soft systolic murmur not transmitted to the axilla while the second is fairly distinct. The second pulmonic is accentuated the second aortic clear. There are no diastolic murmurs. The striking feature is a loud superficial blowing systolic murmur heard over an elliptical area bounded above by the level of the fourth interspace, to the left by a point 9 cm. from the midline, below by the middle third of the ensiform cartilage, and to the right by a point 1 cm to the right of the sternal margin. This represents the tricuspid area. The heart's action is extremely rapid, about 180 per minute, and is irregular, the pulse still more so, as about 40 beats per minute are ineffectual and do not open the aortic valves. The radial pulse is therefore 140 per minute. The right jugular vein is rather full and shows a definite "single" systolic pulsation coincident with the apex beat. This is borne out by the tracing, upon which there are no waves of auricular contraction. The pulse is small, of rather low tension, and very irregular. The vessel wall is not sclerotic.

Blood-pressure with the Erlanger apparatus. Maximal varies from 100 to 110 mm. Hg; minimal varies from 70 to 80 mm.

The liver is much enlarged and extends almost to the level of the umbilicus. Its surface is smooth, the edge round and fairly soft, but it does not pulsate.

His venous tracing is shown in Fig. 74, page 74.

The patient improved somewhat during his stay in the hospital; but his pulse remained rapid, he was bedridden, and died two months later.

DIAGNOSIS.

In the cases in which the pulse-rate is above 160 per minute the diagnosis rarely presents any difficulty, for the tachycardias of simple nervous origin, on the one hand, and those of organic cardiac disease rarely reach that height. But in the border-line case in which the tachycardia is about 140, the diagnosis may be difficult. The crucial point in the differentiation lies in the suddenness of the change of rate, and for this it is important to have observed the beginning and the end of an attack, the sudden rise to maximum rate within a few seconds indicating idiopathic paroxysmal tachycardia, while a gradual step-like or progressive rise indicates a simple tachycardia. Thus while the patient G. R. exemplifies the idiopathic condition, the following case is typical of the simple tachycardia.

Case of Simple Emotional Tachycardia Resembling Idiopathic Paroxysmal Tachycardia.

The patient, a biological student at the University of Virginia, aged 20 years, had recovered from an attack of typhoid fever about a year before. Previously to this he had been strong and free from cardiac symptoms, but since convalescence he is troubled with attacks of palpitation and tachycardia in which the pulse-rate rises from about 60 to about 120 per minute. Slight mental excitement and even the mere mention of taking a pulse tracing suffices to bring on an attack. Before the apparatus could be applied he felt his pulse begin to rise. In the successive quarters of a minute the pulse-rate was 15, 21, 26,

30, having doubled itself within a single minute. But the change of rate was not sudden!

Physiological examination was negative, heart not being enlarged and sounds normal. The case was therefore considered a simple tachycardia. A favorable prognosis was given, which was verified by the subsidence of tachycardia and palpitation within a few months.

TREATMENT.

Drugs.—As regards the treatment of paroxysmal tachycardia various methods have been employed. The first essential is to put the patient into the best possible physical condition, to treat any anæmia, digestive disturbance, constipation, disturbance of vision, heaving, enteroptosis, or other conditions which may bring about reflex irritations; to stop the use of tea, coffee, tobacco, and alcohol, and so to arrange the life of the patient as to do away with mental excitement, worry, over-exertion, and fatigue. If necessary, a "rest cure" may be resorted to. These measures may do much to diminish the frequency of the attacks, but often the latter do not disappear altogether. Small doses of digitalis or aconite may be tried in the interim between attacks, in the hope of keeping them down by increasing the action of the vagus; or potassium bromide or valerian may be given, in the hope of quieting the nervous system; but the desired result is only occasionally obtained.

To quiet the attack after its onset drugs are of little avail. Morphine, bromides, etc., and other sedatives may diminish the intensity of the symptoms, but do not slow the heart-rate. The administration of a few whiffs of chloroform, of amyl nitrite, nitroglycerin, Hoffman's anodyne, strychnine, digitalis and its derivatives (digitalin, digalen), is without effect. Though large doses of strophanthin sometimes stop auricular fibrillation in animals, intravenous injection of strophanthin has not given satisfactory results in three cases of paroxysmal tachycardia to whom it has been administered in the Johns Hopkins Hospital. Aconite also, though the best stimulant for the vagus, was without clinical effect in the one case in which the writer used it.

The application of an ice-bag to the precordium sometimes relieves the symptoms, but only rarely is a sufficient cardiac sedative to stop an attack.

Mechanical Methods for Stopping the Attacks.—A novel method has recently been suggested by Fairbrother based on experiments upon himself. He has found that he can cause his own attacks to stop by any sudden or violent exercise during the attacks, such as running, jumping, skipping rope, etc. Needless to say, these methods, if resorted to in any individual case, should be superintended by the physician and used with the utmost caution.

The paroxysms themselves may sometimes be stopped by various mechanical methods which stimulate the vagi; deep inspirations (Nothnagel), especially yawning, "squeezing arms and elbows tightly against the chest while holding breath and compressing abdomen" (Valsalva's experiment with elbows compressed against chest) (Hay), swallowing, especially of ice-water, or belching, may be successful.

Max Herz has found it possible to suppress many troublesome attacks in his patients by bringing about belching in the following manner: The patient is made to sit down [611]

his mouth with water, bend his head backward as far as possible, and swallow. This not only brings about a desire to belch, but also facilitates the eructation of a large amount of gas and frequently brings the attack to a close. Needless to say care should be taken that the belching does not pass over into continuous air swallowing and that the patient does not acquire this pernicious habit (see page 604).

When belching fails to stop the attack, vomiting may be resorted to, and frequently proves an effectual though unpleasant method. Tickling the pharynx with the finger is usually sufficient to bring it about, especially after swallowing some water. Emetics need not be used.

One of the oldest and best procedures (Bensen, 1880) is pressure upon the vagus just to the left of the thyroid cartilage. The nerve which is just behind the carotid artery is pressed very firmly against the vertebræ and held tightly for two or three minutes. In a considerable number of cases this stops the attack, but in many it fails, or succeeds for a moment and then the tachycardia is resumed (Priesendorfer), just as is the case in the experimental auricular fibrillation.

However, when the results of all methods of treatment have been taken into account, one is inclined to share the feelings expressed by Mackenzie when he wrote. "In my early days I, too, thought I knew how to stop attacks, but more extended experience has shown me that when they stopped it was from some cause unknown to me and which was independent of any means I employed."

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II.

THYROID HEART.

The cardiac disturbances associated with thyroid disease were the most striking features observed by Parry in 1815 in the first described cases of exophthalmic goitre. His first case died of heart failure. Graves (1835), Basedow (1848), Stokes (1854), and Trousseau (1856) were also impressed by the cardiac features of this disease. Trousseau found them especially important in the "*formes frustes*" or "atypical" forms to which he called attention, likening such cases to a defaced ("fruste") coin

The important rôle which these "*formes frustes*" of Basedow's[1] disease play in many cases of so-called cardiac neurasthenia and hysteria has, since Trousseau, been recognized with increasing frequency, and especially since Friedrich Kraus in 1899 called attention to them by introducing the term "Kropfherz" ("goitre heart" or thyroid heart), which is now widely used in Germany.

FORMS OF CARDIAC DISTURBANCE DUE TO THYROID DISEASE.

Strictly speaking, as shown by Rose, Schranz, and Minnich, there is some cardiac disturbance with all forms of goitre. Four main forms of cardiac disturbance may thus be distinguished, due to:

I. Pressure of the goitre upon the trachea, bronchi, veins, chest, and sympathetic ganglia (in simple goitre), pneumo-mechanical goitre heart (Rose).
II. Hypothyroidism (in myxœdema, cretinism, and achondroplasia).
III. Hyperthyroidism (exophthalmic goitre and formes frustes).
IV. Goitre secondary to the cardiac disease (goitre cardiaque, "cardiac goitre").

CARDIAC DISTURBANCES FROM PRESSURE OF THE THYROID.

Potain in 1863 and Rose in 1878 reported cases of heart failure and more or less sudden death in cases in which large colloid goitres pressed upon the veins and trachea. Such a goitre has several mechanical effects:

1. It may prevent adequate filling of the lungs and thus produce emphysema, deficient aeration of the blood, and later asphyxia. The chronically deficient aeration of the blood may lead to secondary cardiac overstrain and finally to myocardial weakness. This will be enhanced by all pulmonary infections.

[1] Basedow ("Base-dō"). Dock after a careful study of priorities advises the acceptance of this name, which was the first unobjectionable term given and is the one most widely accepted

2. The goitre often presses on the sympathetic ganglia on one or both sides of the neck, thus stimulating the accelerators and bringing on a chronic tachycardia just as is produced in Basedow's disease. Müller's muscle in the orbit may also be stimulated and exophthalmos produced. This exophthalmos is often unilateral. The condition of exophthalmos and tachycardia from the pressure of a simple goitre is known as pseudo-Basedow's disease.[1]

CASE OF SIMPLE GOITRE RESEMBLING BASEDOW'S DISEASE.

Such a case is represented by that reported by Potain in 1863.[2] M. K., servant girl, aged 50. Complains of palpitation, feeling of pressure in chest, attacks of suffocation, irregular menstruation. She has had goitre all her life, unaffected by iodine treatment. For some years her eyes have been larger than before. She has lost weight, has suffered from dyspnœa especially on exertion, she has throbbing of the goitre, and her legs are swollen. Her pulse is 152, irregular. Apex is in the sixth interspace 13 cm. from midline. There is heaving of the entire precordium. At the apex and over the precordium there is a meso-systolic murmur. The veins of the neck are dilated. There is a large goitre which does not pulsate and no murmur is heard over it. Digitalis is without effect, and the patient died from pulmonary œdema 11 days after admission.

Autopsy showed colloid cystic goitre with some hemorrhages from ataxia, slight infarction of the lungs, and a somewhat enlarged, very flabby heart.

The livor, orthopnœa, asphyxia, and sudden death, as in Rose's case, are due to pressure upon the air-passages, and are to be regarded as cardiac symptoms. The respiratory origin of this suffocation is seen in the very marked inspiratory (not systolic) retraction of all the thoracic interspaces.

CARDIAC AFFECTIONS OF HYPOTHYROIDISM (CARDIOPATHIA THYREOPRIVEA) (KRAUS).

In all the conditions in which there is atrophy of the glandular tissue of the thyroid and diminution in the internal secretion of the gland, there are symptoms of cardiac weakness. The patients get out of breath on very slight exertion. The pulse is small and weak, but may be either slow or slightly accelerated (Kraus). This is due to the fact that the physiological vagus tone is largely due to the thyroid secretion (v. Cyon), and when it is deficient there is an overstimulation of the accelerators. However, as Kraus points out, the cardiac features in cachexia thyreoprivea are not prominent features of the disease, and hence are of little importance in connection with diseases of the heart.

Revilliod has, however, called attention to another effect of hypothyroidism upon the circulation, namely, early arteriosclerosis with calcareous deposits. This effect has also been produced by v. Eiselsberg in new-born lambs from which he removed the thyroid glands. In contrast to other experimental arteriosclerosis, the arterial changes affected the intima and not the media.

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[1] In some cases, however, this is not due to pressure on the sympathetic but to the activation of thyreoglobulin by the iodine treatment. Occasionally, moreover, a goitre shows in one part colloid degeneration, in another hyperplasia like that of Basedow's disease.

[2] Bull. de la Soc. d'Anat. de Paris, 1863, p. 87, quoted from Minnich.

DISTURBANCES DUE TO HYPERTHYROIDISM.

Basedow's Disease (also "Formes frustes," and Accidental Hyperthyroidism in the Treatment of Obesity).

As stated above, these conditions present the most important cardiac features which are due to disturbed thyroid metabolism.

PATHOLOGY, PATHOGENESIS, AND PATHOLOGICAL PHYSIOLOGY.

The veil of mystery has been lifted from diseases of the thyroid by the hands of the physiological chemists. The surgeons Astley Cooper, Reverdin, and Kocher had found that extirpation of the thyroid for goitre led to myxoedema, and Pisenti Tiley and Vassale had demonstrated that these symptoms could be prevented by feeding the dried thyroid substance. But the accurate knowledge dates from the studies of Baumann and his pupils, Roos and Oswald.

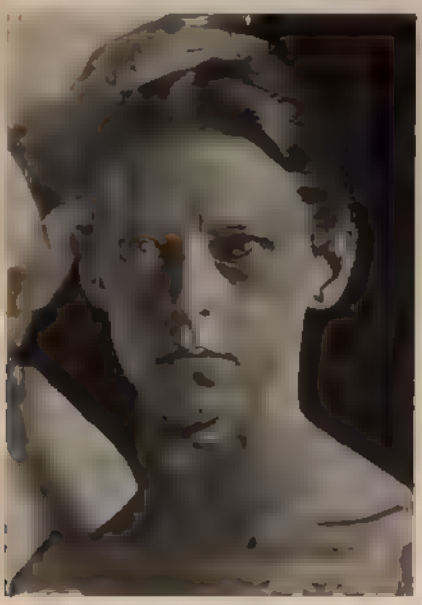


FIG. 321.—Photograph of a patient with Basedow's disease. (Kindness of Prof. Bloodgood.)

Thyreoglobulin and Iodothyrin.—Baumann, Roos, and Oswald have shown that the active principle of the thyroid is a globulin (iodothyreoglobulin) which contains all the iodine of the gland. Thyreoglobulin is at first formed within the cells free from iodine and later acquires its iodine from the blood, becoming iodized thyreoglobulin or iodothyreoglobulin. The cells secrete thyreoglobulin more readily after it is combined with iodine. In cases of colloid goitre when the blood content is low in iodine, the cells become loaded with the iodine-free thyreoglobulin and undergo colloid degeneration. Iodine-free thyreoglobulin is physiologically inactive, and the entire activity of the gland is due to the iodized thyreoglobulin. Indeed, as Baumann has shown, it is due to a comparatively simple molecular group with which the iodine is combined, and which can be split off from the rest of the globulin molecule by hydrolysis with H_2SO_4 (iodothyrin).

Effect of Thyreoglobulin on Thyroid Structure.—According to Oswald, it is the state of the thyreoglobulin which determines the histological changes in the thyroid. When the iodine-free thyreoglobulin accumulates in the cells, they become overloaded with colloid and gradually undergo colloid degeneration, so that the acini are found surrounded with the original single layer of flat epithelial cells in all stages of colloid degeneration, whose disintegration adds to the colloid within the lymph spaces and within the acini. (Oswald, Huerthle.)

An excess of the iodized product, on the other hand, stimulates the cells to hyperplasia, so that instead of a single layer of columnar epithelium the cells about the acini are found to be several layers and protrude into the lumen in irregular papillary masses suggesting adenomatous changes (Halsted, Oswald, MacCallum, Wilson). The same hyperplasia takes place as a compensatory process when a part of the gland is removed (Halsted, Marine). The colloid gradually disappears from the lumen as glandular activity and hyperplasia progress and as the symptoms become more

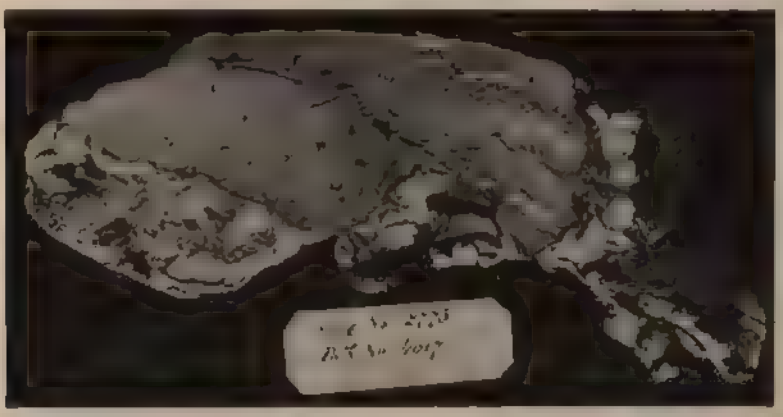


FIG. 322.—Photograph of a portion of the thyroid gland removed from the patient shown in Fig. 321. Kindness of Prof. Bloodgood.

severe, and in very bad cases it may be entirely absent (Marine and Williams, Wilson). The arteries and veins are very much dilated (C. Gerhardt). When the iodine is administered in cases of colloid goitre, the excess of thyreoglobulin may be suddenly iodized and by escaping into the blood may give rise to symptoms of hyperthyroidism (palpitation tachycardia, tremor, loss of weight, exophthalmos—Basedowification of a simple goitre). When there is an excess of iodine and iodized thyreoglobulin in the blood, the symptoms are the same as arise from the administration of thyroid substance (thyreoglobulin or iodothyrin, its split product).

PHYSIOLOGICAL EFFECTS OF THYROID SECRETION.

It has been shown that the effects of excess of thyroid secretion in the blood are:

1. An increase in metabolism, especially in the oxidation processes and the breaking down of proteids in the tissues and bone, giving rise to an increase in N and P_2O_5 (Fr. Muller) excreted and in the gas metabolism (Magnus-Levy). In man this finds its concrete expression in the loss of weight, due especially to loss of muscle substance (Baumann and Roos).

2. There is a general stimulation of the peripheral nerves both medullated and sympathetic. V. Cyon, Roos, Oswald and Kraus, and Friedenthal have shown that these substances have several distinct actions on the circulation.

A They stimulate the depressor or afferent nerves from the heart, giving rise on the one hand to the cardiac sensations, palpitation, and anginal pains, and on the other hand to the vasodilation and low diastolic blood-pressure which are often observed in these cases.

B They stimulate both the vagi and the accelerator nerves. The action upon the accelerators predominates, however, and tachycardia is thus produced. The vagus still remains irritable however, and the heart can be slowed by pressure on it. The same stimulation of the other fibres of the cervical sympathetic gives rise to the peculiar ocular signs of Basedow's disease (see page 583).

C Cleghorn has shown that thyroid extract has a direct action on the cardiac muscle, increasing the size and force of the contraction, which manifests itself in the increased pulse-pressure, increased maximal pressure, and cardiac hypertrophy.

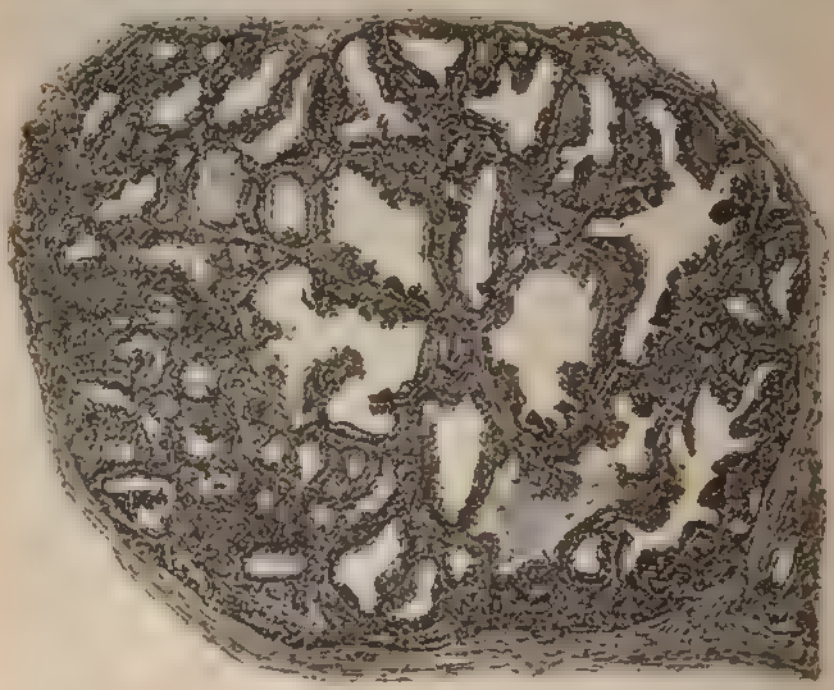


FIG. 323.—Drawing of a histological specimen from the same thyroid. The histological picture of advanced thyroid hypertrophy observed in cases of Graves's disease in which the symptoms are most marked. (Bloodgood, Surg. Gyn. and Obstet., August, 1905, vol. i, p. 113.) This drawing was made in June, 1903.

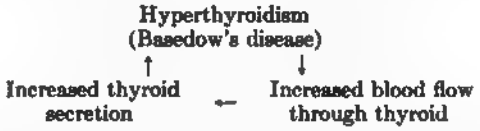
Biochemical Evidences of Hyperthyroidism.—Falta and Zuelzer, Kraus and Froelenthal have shown that thyroid extract directly antagonizes adrenalin in its pupillo-dilator action on the frog's eye, and that this can be used as a test for hyperthyroidism in clinical cases.

Another important biochemical blood test for hyperthyroidism is that of Reid Hunt, who has shown that the blood of such patients increases the resistance of mice to poisoning with acetonitrile and morphine, so that the lethal dose is thus doubled.

All these investigations have proved without doubt that in Basedow's disease there is an excess of thyroid secretion into the blood (as claimed by Möbius) and that the secretion is indistinguishable from that of the normal thyroid, representing a condition of hyperthyreosis (increased secretion) rather than of dysthyreosis (altered secretion). Whether they will be of practical value in the diagnosis of the puzzling "formes frustes" remains still to be determined, since the excess of thyreoglobulin in the blood of these cases may be too small for chemical recognition.

It is possible that this anti-adrenalin action may be responsible for the brownish pigmentation (Jellinek's sign) which occurs in many cases of hyperthyroidism, especially about the eyelids. This pigmentation somewhat resembles the pigmentation of Addison's disease (lack of adrenalin secretion). Kraus and Friedenthal have also found that this antagonistic action upon the frog's pupil is valuable in diagnosis, since it is given by the blood of patients with Basedow's disease, but not by the blood of neurasthenics and hysterical patients.[1]

V. Cyon has shown the very important fact that injection of thyroid exact or iodothyrin causes an increased blood flow through the thyroid gland, probably thus acting as a hormone to increase its own secretion and to introduce a vicious circle:



It is this increased dilatation of the arteries which gives rise to the murmurs over the thyroid in Basedow's disease (Guttmann).

ETIOLOGICAL FACTORS.

Basedow's disease is more common in women than in men (805 women, 175 men in Buschan's 980 cases); 60 per cent. occur in the fourth decade of life (Buschan). In Passler's 58 cases there were 4 under 15 years, 29 at from 15 to 25 years, 18 at 25 to 45 years, 7 over 45 years.

Basedow's disease is very widespread, but is somewhat more rare in regions where simple goitre is common than elsewhere, perhaps owing to the lesser intake of iodine. Heredity plays some rôle; mental and nervous disease, diabetes, and tuberculosis are often found in the same family. In one famous family reported by Osterreicher eight out of ten children of a hysterical woman had Basedow's disease, and one of these daughters had three children with the same illness.

The following list gives the predisposing factors in the series of A. Kocher and of Landström:

	Cases.
Gradual onset with etiological factors unknown	28
Pregnancy	10
Chlorosis	7
At first menstruation	6
After fright, shock, or grief	5
After fatigue	8
Infectious diseases (influenza alone, 7)	13
Old simple goitre	5
Sojourn at high altitude	2
Heredity	1
Appendicitis	1
Total	86

[1] It seems doubtful whether the blood of cases with mild formes frustes contains enough excess of thyreoglobulin to give this test a hard-and-fast diagnostic significance.

It will be seen that infectious diseases and especially influenza constitute the most common cause. De Quervain has found a subacute thyroiditis quite common in these conditions, especially in influenza, typhoid fever, rheumatism (as in Parry's first case), and diphtheria, and this thyroiditis was followed by Basedow's disease within a few months in about 20 per cent. of the cases. Boggs and Sladen have found mild thyroiditis present in most of the cases of typhoid fever in which the pulse is over 120 at the height of the fever. Tonsillitis may also be a forerunner, and Engel-Reimers has found acute thyroiditis in secondary lues leading to Basedow's disease. After pregnancy the hyperthyroidism which is normally present in that condition may increase and lead directly into Basedow's disease. The coexistence of puerperal infection, mastitis, fright, grief, or shock undoubtedly predisposes to the disease, as in the case of a girl under Friedrich Muller's care, whose symptoms began when she was suddenly deserted by her lover just after the birth of an illegitimate child. In one of v. Graefe's cases the symptoms set in within a few days following a night of sexual excesses. These factors may act by producing a reflex dilatation of the vessels in the thyroid. Thus, Trousseau writes of a woman of 53 who suffered deep grief from the death of her father. "One night, after she had been crying for a long time, she suddenly felt her eyes swell and lift up her lids, her thyroid gland increase notably in size and throb in an unusual manner; she had at the same time violent palpitation of the heart." The writer on one occasion had the opportunity to observe a case of acute enlargement of the thyroid in a man of thirty, associated with tremor, tachycardia, palpitation, slight v. Stellwag's but no other ocular sign. The disturbance followed the ingestion of two cups of strong coffee at a time of great worry and was complicated by a mild attack of "grippe." The enlargement of the thyroid was sufficient to prevent buttoning the collar. It subsided entirely after 24 hours, and with it the symptoms of hyperthyroidism. It is probable that the grippe (influenza or streptococcus infection) rendered the thyroid particularly sensitive.

SYMPTOMS.

The classical pathognomonic symptoms of exophthalmos are the well-known triad of struma, tachycardia, and exophthalmos, or the tetrad of struma, tachycardia, exophthalmos, and tremor.

These are well described by Parry (1815) in his first case: a married woman, aged 37, who had caught cold in lying in, and for a month suffered under a very acute rheumatic fever. Subsequently she became subject to more or less palpitation of the heart very much augmented by bodily exercise and gradually increasing in force and frequency till my attendance, when it was so violent that each systole shook the whole thorax. Her pulse was 196 in a minute, very full and hard alike in both wrists, irregular as to strength, and intermitting at least once in six beats. Twice or thrice she had been seized in the night with a sense of constriction and difficulty in breathing, which was attended with spitting of a small quantity of blood. She described herself also as having frequent and violent stitches of pain about the lower part of the sternum. About three months after lying in, while she was suckling her child a lump about the size of a walnut was perceived on the right side of her neck. This

continued to enlarge till the period of my attendance, when it occupied both sides of her neck so as to have reached an uncommon size, projecting forward before the lower angle of the jaw. The part swelled was the thyroid gland. The carotid arteries on both sides were greatly distended, the eyes were protruded from their sockets, and the countenance exhibited an expression of agitation and distress, especially on any muscular exertion, which I have rarely seen equalled. Bowels were usually lax. . . . For a week she has had œdematous swelling of her legs and thighs." (The patient died with symptoms of heart failure.)

Besides the pathognomonic triad, increased nervous excitability, tremor, loss of weight, and pigmentation of the skin, especially about the eyelids, are important accessory symptoms.

The chief symptoms of Basedow's disease may be grouped in the following categories, and arranged in what is approximately the order of increasing severity.

Cardiac Phenomena. — Palpitation, continuous slight elevation of pulse-rate, with occasional attacks of intense tachycardia brought on by emotion, excitement, or exercise, or occasionally on awakening; visible pulsation and dilatation of carotid arteries; pulse collapsing, angina pectoris; hypertrophy of the heart, precordial heaving and intense pulsation; irregularity of pulse; dilatation of heart; heart failure; ascending œdema, etc.

Psychic Symptoms. — General nervousness, insomnia, restlessness, mental exuberance alternating with depression and melancholia, delusions and hallucinations.

Ocular. Staring gaze without winking for considerable periods. Widening of palpebral slit (Dalrymple, v. Stellweg's sign), lids do not follow eyeballs perfectly, a white streak of sclera is seen between lid and cornea, especially on glancing downward or upward (v. Graefe's sign), inability to converge in looking at near objects (Möbius' sign), exophthalmos, overflow of tears, pain and feeling of tension in the eyeballs, corneal ulceration.

Peripheral Nerve Symptoms. — Fine tremor (from 8 to 10 per second), especially of the finger tips, nystagmus, superficial and cog-wheel breathing, astasia-abasia, hyperæsthesias and paræsthesias occasionally, inability to frown or wrinkle forehead (Joffroy's sign).

Cutaneous from vasodilation and anti-adrenalin action). Feeling of heat, continuous and intense, lowered electrical resistance; sweating, color usually pale brownish — Addison like pigmentation, especially about eyelids (Jellinek); flushes, localized transitory œdema, especially about eyelids; scleroderma.

Nutritional (increased rapidity of metabolic processes; loss of N and P_2O_5). — Loss in weight; sometimes absolute anorexia, sometimes excellent appetite; attacks of diarrhœa, often with slimy stools; polyuria, glycosuria. Fever (varying from 99° to 104°).

Blood. — Slight leucocytosis without change in red blood-corpuscles or secondary anæmia; polymorphonuclears 50-55 per cent., lymphocytes 20-25 per cent., large mononuclears 8-16 per cent. (large mononuclear leucocytosis present in formes frustes). (Barker, Caro.)

Psychic Manifestations.—The psychic symptoms in hyperthyroidism have been very aptly compared to the well-known effects of over-indulgence in coffee,—increased activity of thought, restlessness, irritability, insomnia, and in the more severe cases garrulity and delusions. As mentioned above, over-indulgence in coffee may sometimes be followed by enlargement of the thyroid. There can be little doubt that many cases of so-called neurasthenia and hysteria are due to a more or less transitory state of over-secretion of the thyroid. This is particularly true when the symptoms are accentuated at the menstrual periods, for then the thyroid secretion is increased. It is possible that, as suggested by Graves, the "globus hystericus" may be due to an acute swelling (erectile expansion) of the thyroid. Neurasthenic symptoms may, however, have a basis in hyperthyroidism in cases when this would be least expected. For example, a young physician in robust health recently complained to the writer of having suffered from insomnia and palpitation for several months, during which time he had been compelled to forego his accustomed daily exercise. On closer observation, however, he observed that at about the time his symptoms had begun he noticed a slight swelling of his thyroid which had persisted ever since, although he had no tremor.

Cardiac Signs and Symptoms.—The *cardiac symptoms* also have some similarity to those of an overdose of coffee, especially the palpitation. This symptom is probably due to the direct stimulation of the afferent nerves of the heart (depressor), which has been shown by v. Cyon to result from injection of thyroid extracts, iodothyroin and thyreoglobulin. Palpitation is the earliest and often the most severe symptom.

The *tachycardia*, like most of the signs of Basedow's disease, results from the stimulation of the accelerator nerves and from the degree to which this outweighs the effect upon the vagus. The pulse-rate may be continuously elevated (over 120), or the tachycardia may be latent and attacks of rapid pulse may be brought out only by slight disturbance of the equilibrium or by the administration of very minute doses of thyroid extract (Emerson, quoted by Barker). In these attacks the *pulse-rate rises gradually* during a few minutes and *falls gradually* (in contrast to idiopathic paroxysmal tachycardia), but in one case v. Hoesslin has seen sudden doubling and sudden halving of the rate. Strubing has found that pressure upon the vagus slows the rapid heart of Basedow's disease, showing that there is no paralysis of that nerve.

Although, as Cleghorn has shown, *thyroid extract increases the force and size of cardiac contraction* (the increased pulse-pressure shows increased cardiac output), the persistent over-stimulation of this organ draws so much upon its reserve force that it may readily suffer from overstrain and undergo acute dilatation. Afferent impulses through the depressor nerves, which are already in a state of increased irritability, may give rise to symptoms of typical angina pectoris, with referred pain down the arms and precordial hyperæsthesia. This thyroid type of angina pectoris has been described on page 293. Prolonged overstrain may result in failure of either the left or the right heart, and symptoms of pulmonary or systemic decompensation (œdema, ascites, etc.) set in.

The irregularity is probably due to occasional extrasystoles, though careful analyses of its nature are lacking. In one case reported by v. Hoesslin there was definite paroxysmal tachycardia with sudden onset and sudden cessation—approximate halving and doubling of rate, but Hirschfelder finds that this condition is a rare one in Basedow's disease. The attacks of tachycardia and palpitation most commonly begin and end by a gradual, though rapid, change of rate, and indicate a simple exaggeration of physiological variations.

The maximal blood-pressure is usually high, the minimal normal; the pulse-pressure increased; this shows that there is an increased systolic output with low peripheral resistance, and corresponds well with the experimental results from injection of thyroid tissue juice (Pressaft). In 10 cases of Basedow's disease Krause and Friedenthal found

	Lowest		Average		Highest	
	Cm. H_2O.	Mm. Hg.	Cm. H_2O	Mm. Hg.	Cm. H_2O	Mm. Hg.
Maximal blood-pressure. .	115	106	182	134	215	158
Minimal blood-pressure	85	62	89·5	65·8	90	66

This accords with the writer's experience, but in the early cases and "formes frustes" the maximal pressure may not be elevated even when there is tachycardia.

The heart is usually enlarged and hypertrophied, the apex impulse forcible, and the large systolic excursions impart a heaving to the whole chest. In periods of overstrain from exertion or excitement there may be transitory dilatation of the heart, and this uniformly occurs during the chronic heart failure. There is often a blowing systolic murmur heard over both ventricles and at the apex, perhaps due to functional insufficiencies of the auriculoventricular valves.

Heart failure is the immediate cause of death in most cases of Basedow's disease.

Ocular Manifestations.—The ocular manifestations are peculiar and very characteristic. V. Graefe (1857) called attention to the fact that when the eyes moved upward and downward the lids did not follow them perfectly, but a streak of white sclera could be seen between lids and cornea (Graefe's sign). Dalrymple and in 1867 v. Stellwag noted the widening of the palpebral slits, the staring expression, the absence of winking. V. Stellwag's sign is in most cases the earliest characteristic sign of Basedow's disease. Mobius' sign is the inability to converge the two eyes when looking at a very near object.

The origin of these signs is very simple. Claude Bernard, when he first stimulated the cervical sympathetic, demonstrated that widening of the palpebral slit and dilatation of the pupil resulted and that the eyeball was pushed forward. Aran and Kaufmann (1860) demonstrated that this exophthalmos resulted from stimulation of Muller's non-striated muscle in the eyelid, which is innervated by the cervical sympathetic. These experiments were confirmed by a number of writers, especially MacCallum and Cornell (1904). The exact course of the fibres of Muller's muscle and their mode of operation has been described by Landstrom. Landstrom finds that the fibres of smooth muscle form a narrow cuff, or truncated cone, encircling the anterior portion of the orbit. The fibres at the posterior border of the cuff pass backward and are inserted into the sclerotic coat of the eyeball. The fibres forming the anterior margin of the cuff are inserted into the

upper or lower lids, in which they run obliquely toward the palpebral slit. The middle portion of the cuff constitutes the fixed point from which the muscle acts, and is attached by short fibrous bands to the bony wall of the orbit. Contraction of this muscle therefore tends to draw the eye forward (exophthalmos) as well as to pull the lids apart (Dalrymple and v. Stellwag's sign). The delicate coordination of lid movement and eye movement is disturbed by this added traction upon the lid (v. Graefe's sign). Moreover, the contraction of these fibres tends to keep the axes of the eyes divergent, and thus antagonizes convergence (Möbius' sign).

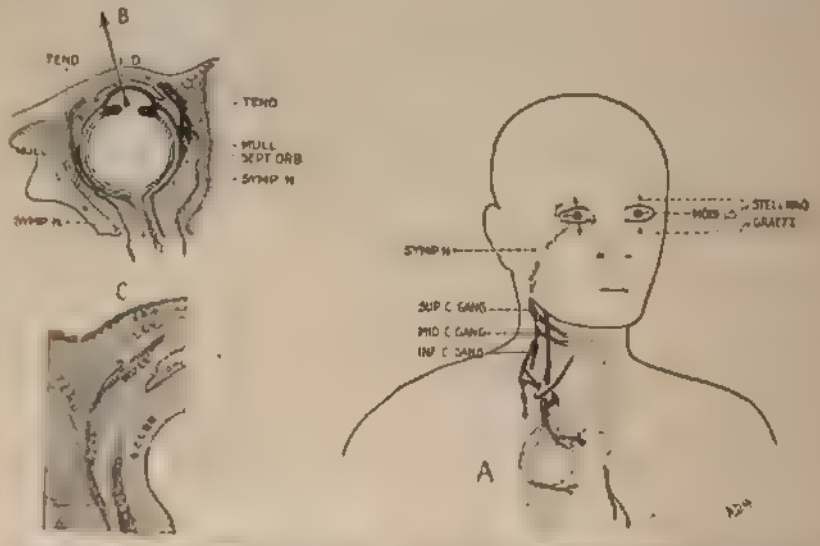


FIG. 324.—Diagram showing the relation of the various anatomical structures concerned in the production of the ocular and cardiac manifestations of Basedow disease. A, Distribution of the branches of the cervical sympathetic to the heart, thyroid gland, and eyelid. The arrows indicate the direction in which stimulation of the cervical sympathetic excites the eyeball and eyelid. *SYMPN*, sympathetic nerve plexus; *SUP C GANG*, *MID C GANG*, *INF C GANG*, superior, middle, and inferior cervical ganglia. B, Relation of Müller's muscle to the eyeball and structures within the orbit (schematic). Müller's muscle (*MULL*) is shown in black. The arrows indicate the direction of its pull. *TEND*, tendinous attachment of Müller's muscle to the orbit, septum orbitale (*SEPT ORB*). C, Section through the lateral portion of the orbit (semi-schematic, modified from Landström). *ORB*, orbitalis; *LEV*, levator palpebrarum; *CONJ*, conjunctiva; *SCLER*, sclera; *RECT MED*, rectus medius.

Muscular Changes.—A fine tremor beginning in the fingers, with 8–10 contractions per second, has been shown by Marie to be almost common enough to be included among the cardinal symptoms. It is probably due to the overstimulation of the peripheral nerves, and finds its analogue in the tremor from coffee and tobacco. Tremor of the tongue and sudden movements of the tongue and lips are not as common as in alcoholism. It is probable that the muscular weakness consequent upon the katabolism of muscle proteid aids in the tremor.

Astasia-abasia (giving way of the legs in standing and walking) is rare, but has been reported. It represents an extreme grade of nervous disturbance.

The increased metabolism of N and P_2O_5, with destruction of muscle tissue, fat, and to a lesser extent of the bones, is important and finds its expression in the general loss of weight (often 25 to 50 pounds). It is the direct result of iodothyrin intoxication.

Diarrhœa is common. There is often a good deal of mucus in the stools, suggesting some relation to the so-called mucous colitis.

Changes in the Thyroid itself.—As regards the size and appearance of the thyroid gland there is great variation. In spite of the common term of "exophthalmic goitre," the thyroid may not be prominent nor even palpable. Since there is great variation in the average size and weight of the thyroid in different regions, 25 to 33 Gm. in certain regions, 60 Gm. in others, 100 Gm. in Switzerland (Oswald), —a merely palpable thyroid need be of no diagnostic importance. Increase in the size of the thyroid is equally difficult to interpret. The size of the thyroid bears a definite relation to sexual activity, and increases regularly during menstruation and pregnancy, often to a considerable degree. Indeed, in some cases of formes frustes it is not unlikely that we are dealing with slight hyperthyroidism whose intensity is determined by these physiological factors. Increased vascularity is of great importance in differentiating between transitory and persistent hyperthyroidism. It can be demonstrated by eliciting a murmur and thrill over the thyroid when the gland is pressed upon (Guttmann). This cannot be produced in simple goitres or normal glands.

SECONDARY HYPERTHYROIDISM.

Moreover, it is probable that in many neurotic, toxic, and organic diseases the actions of nerves or of hormones arouse the thyroid to a secondary activity, which may, nevertheless, be of great importance in determining the features of the case. For example, Holz has reported two cases of exophthalmic goitre in children in whom the disease subsided on removal of the adenoids; one case recurred and again subsided with the recurrence and removal of the adenoids. Accordingly it is advisable not only to treat the Basedow's disease but also to look for and treat the other foci of excitation.

DIAGNOSIS.

It is evident that, though there can be little doubt as to the nature of well-developed thyroidism, there may be room for much debate regarding cases of formes frustes, for these cases must be differentiated from simple physiological hypoactivity of the thyroid. Patients should be carefully watched for the development of ocular signs, especially at menstruation, since these are practically never present in persons whose thyroid activity is normal.

In cases in which symptoms are so mild, however, it is still important to bear in mind the possibility of a thyroid origin for the condition at least in so far as an increased thyroid secretion may arise reflexly and perpetuate itself through the vicious circle mentioned on page 579. It is probable that on this basis the origin of many an obscure "cardiac neurosis" will be cleared up. Hyperthyroidism and hysteria, sexual neurasthenias, epilepsy, tobacco poisoning, alcoholism, myocardial disease, and valvular diseases are frequently associated, and when one of these conditions is present it still remains important to look out for contributing rôles on the part of the thyroid.

Each case of morbus Basedowii may be considered as an autointoxication due to the passage of more or less iodized thyreoglobulin from the thyroid gland into the blood. When this is secreted in large quantities, the condition is outspoken and presents many of the symptoms, among them some of severe grade. When but little excess of thyreoglobulin circulates in the blood, it may give rise to the "formes frustes" with but few symptoms and those of the milder type predominating.

However, even in the most atypical cases of "forme fruste" one or more of these symptoms may reach excessive severity, and the disease may persist in the form of a cardiac neurosis, a psychosis, a chronic enteritis, a progressive inanition, a diabetes, or even a mild relapsing fever, for long periods. The cardinal suggestive signs may be so slight in intensity as to be noticed only when the suspicion of Basedow's disease has once been aroused in the mind of the examiner, and then the coexistence of several unobstrusive features may make the condition definite; as, for example, a slight staring, anxious expression in a thin, nervous woman who suffers from attacks of palpitation and precordial pain and who manifests a slight fine tremor of the fingers and a tendency to diarrhœa. On closer examination it may be found that the lids do not follow the eyeballs perfectly and there is slight fulness of the neck, but none of these symptoms are striking.

Case of Basedow's Disease with Anginal Attacks.

Mrs. K. M., housewife, aged 23, seen under treatment at the Johns Hopkins Medical Dispensary on Dec. 29, 1906, when she complained of palpitation of the heart and pain in the right chest going down the arm. She is quite nervous and sometimes has crying spells.

She is a rather pale woman, fairly nourished. The gums and mucous membranes are a trifle pale. The palpebral slit is wider than normal, but lids follow eyes. Convergence is, however, not perfect. The outlines of the thyroid gland can be seen; the gland is readily palpable, but not much enlarged. The lungs were clear on auscultation and percussion.

The heart was not enlarged; sounds clear. Pulse of good volume, regular in force and rhythm, blood-pressure apparently low.

She was given Blaud's pills and also tincture of aconite 0.3 cc. (m v) and potassium bromide, without relief. She was seen a number of times during the course of the next year, during which she passed through a normal pregnancy and labor. Palpitation continued. A well-marked exophthalmos developed and palpebral slits became a little wider than normal.

During April, 1907, she had attacks of pain over the left side of the chest and down the front (extensor surface) of the left arm, sometimes radiating to the shoulder. During attacks there is often tenderness in the fourth left interspace, sometimes also in the fifth, about the mammillary line. It never radiates to the right of the midline. These attacks are accompanied by palpitation and the heart-rate is rapid. She also has a peculiar fluttering sensation, and occasionally an irregular beat. Tracings at this period showed normal venous and carotid pulse. She was given small doses of ergotin without relief. A week later she was given calcium lactate 0.6 Gm. (gr. x) after meals, after which she began to feel better at once, though never relieved by any other medicine. The remedy was, however, far from specific, and the old symptoms returned in spite of the calcium lactate. During the course of the next six months various remedies were given, none of them with marked effect. It seemed to both patient and physicians, however, that she experienced a distinct improvement in symptoms whenever calcium lactate was given and distinct retrogression when other drugs were substituted. Operation was advised but not consented to, and the patient was lost sight of.

ACUTE BASEDOW'S DISEASE SIMULATING MALIGNANT ENDOCARDITIS.

One group of cases to which attention should be especially directed are those of very acute Basedow's disease with fever, prostration, tachycardia, profuse sweats, sometimes chills and slight jaundice—a clinical picture very closely simulating acute endocarditis (W. G. Thompson). These cases are rather rare, but very grave. The diagnosis depends upon the cardinal symptoms aided by a negative blood culture.

PROGNOSIS AND TREATMENT.

Statistics regarding the mortality of Basedow's disease vary considerably, as shown by the following list.

V. Dusch	12.5 per cent.
V. Graefe	12 per cent.
Mackenzie	12.5-25 per cent.
Cheadle	9.6 per cent.
Billingham	18.1 per cent.
Gaill	21.3 per cent.
Charcot	25 per cent.
Buschan (900 cases)	12.5 per cent.
Thompson	10 per cent.
Williamson	25 per cent.

These figures err, on the one hand, because only the serious cases reach the literature, and, on the other, because most of the cases have been followed for only short periods. Williamson, who followed his cases for some years and found a 25 per cent mortality, probably approximates the truth.

J. Berry gives the following statistics of 56 cases treated without operation:

Complete recovery	10
Considerable improvement	24
Little or no change	8
Fatal	14

Even after recovery recurrence is the rule, so that as excellent an observer as August Hoffmann states that in 23 outspoken cases he has not seen a single permanent recovery!

It is evident, therefore, that at the onset of undoubted Graves's disease therapeutic interference is necessary. The best principles in inaugurating treatment are those which may be deduced from the findings of v. Cyon's experiments,—*i.e.*, that the clinical manifestations are due to hypersecretion of thyreoglobulin, that this is proportional to the blood flow through the thyroid, and that the thyroid secretion in the blood tends itself to increase this flow and to produce a vicious circle.

The first essential of any palliative treatment, therefore, is to reduce the thyroid secretion to its lower ebb by the removal of the two stimulating causes—exercise and excitement. In the mild cases a simple isolation cure, with absolute rest in the horizontal position, can sometimes so lessen the flow through the thyroid and the secretion of this gland by diminishing the size and number of heart-beats that the thyreoglobulin content of the blood falls to normal and symptoms subside. If the rest cure be prolonged,

the slight glandular hyperplasia of early cases may subside and a permanent cure may result.

Various measures assist this process, especially those which act as psychic sedatives. Psychotherapy and suggestion, in so far as they tend to lessen the elements of worry, quiet the patient's mind, and thus quiet his heart's action, may aid in tiding over a period of not too intense excitement. Similarly Möbius, the apostle of serum therapy, reports the cure of one case by hypnotism! These are, however, exceptional. Psychotherapy in Basedow's disease is to be classed among the valuable sedative measures, but not among those of fundamental therapeutics. Cold wet packs, especially before retiring, may be of considerable assistance (Eichhorst), as also the bromides and the soporifics (veronal, trional, etc.), though to a less degree. Calcium salts are often very satisfactory as sedatives. The iodobromide of calcium was used by Guptill (1874). In one case (K. M.) under the writer's care calcium lactate was the only drug which caused any symptomatic relief, but even this was not marked. Müller and others have used quinine, especially as the hydrobromate, but in many cases it is without effect.

Iodine as used by the earlier observers may sometimes exert a positively harmful influence by activating (iodizing) still more of the thyreoglobulin, and it may thus bring on an exacerbation of the condition. The effect of potassium iodide is less certainly harmful and is sometimes beneficial, but its action is uncertain.

Galvanization. — One of the oldest and best forms of treatment is galvanization of the cervical sympathetic, with the anode over the carotid artery and the cathode at the nape of the neck. With currents of 2-3 milliamperes, as used by Chvostek, Benedikt, Cardew, and others, it uniformly gives a certain degree of improvement, without effecting a cure. In early cases J. O. Hirschfelder has obtained complete subsidence of symptoms in a considerable number of patients by the use of strong currents (20-30 milliamperes), the negative pole being applied over the sympathetic at the neck, the positive over the thyroid for two or three minutes. After this it is applied over the heart. This vigorous treatment seems to be the best method of applying electricity, but must be continued for several months.

X-Rays. — Exposure of the thyroid to the Röntgen rays was introduced by Pusey, Boggs, and Beck in America, and has had in the main a favorable action. Schwarz (1908) collected reports of 10 cases, showing gain in 26, improvement in nervous symptoms in 40, exophthalmos better in 15, but struma lessened in only 8.

Specific Sera.—Two forms of so-called specific sera are also in use: (1) anti-thyreoidin (thyroidectin), the serum of thyroidectomized sheep (Möbius), has been in use for some years, and in spite of numerous favorable reports has been found absolutely without effect by Ewald, Mackenzie, and Strumpell. (2) Beebe has prepared an antiserum for the nucleoproteid of the thyroid gland from animals into which the purified nucleoproteid thyreoglobulin had been injected, in the hope of bringing about retrogressive changes in this gland. This serum has been used therapeutically by Rogers and by W. G. Thompson, who report distinctly favorable

results, especially in the very acute cases (90 cases: 23 cured, 54 improved, 11 failed, 4 died); but other observers state that the results are no better than those in ordinary hospital practice, and further confirmation is needed.

Operative Treatment.—Thyroidectomy.—The physiological indication for therapy in Basedow's disease is to lessen the amount of thyreoglobulin secreted into the blood. If the various methods intended to affect the gland as a whole are unsuccessful, the secretion may be diminished by removing a large portion of the gland (thyroidectomy). This operation was first successful in the hands of L. Rehn (1881), and has now come into quite general use, especially through the work of Mickulicz and the Kochers in Europe, and Halsted and the Mayos in America.

The operation should be done under local cocaine anæsthesia. It may vary from ligature of the arteries to one-half of the gland, or this may be combined with excision of the latter, or, on the other hand, one-half of the gland may be excised and the arteries supplying a portion of the other may be ligated. The technic and results in large series of cases have been reported by A. Kocher, Landström, and C. H. Mayo, and many of the important details by Halsted and Evans.

Kocher (1907) especially calls attention to the necessity of suiting the extent of the operation to the condition of the patient, especially the cardiovascular condition. "A systolic blood-pressure, even of 195 mm. Hg, does not forbid operation; . . . but if we find the blood-pressure below normal and the disease highly developed, we must study the condition and especially note the action of the heart after exercise or excitement. Under these circumstances we might find a sudden, very marked dilatation of the heart, irregularity of pulse, and a blood-pressure which cannot be measured by our ordinary methods."

The patient should be given a preparatory period of rest and palliative treatment to prepare her for the operation, and two or more operations should be done on the same patient rather than too extensive an operation at one sitting. Kocher never ligates more than two arteries nor removes more than one-half the gland at one sitting, but these measures suffice in cases that are not too far advanced. Halsted has called particular attention to the need of preserving the parathyroid gland in order to avoid tetany. Hence he advocates tracing out the branches to these small bodies and then ligating the main artery beyond them. Both Kocher and Halsted insist upon the greatest care in the ligation of all bleeding points during the operation and in draining off any small collection of serum which may collect during the healing of the wound. This greatly diminishes or obviates the intensification of Basedow symptoms which sometimes result a few days after operation (probably from absorption of iodothyreoglobulin upon the raw surface of the gland) and which may be dangerous. Halsted also found that the continuous use of an ice-bag upon the neck during a few days after the operation retards the absorption from the gland and lessens the frequency of these symptoms.

As a result of this procedure in 254 patients (2 operations in 71 cases), A. Kocher has obtained great improvement in every case, with absolute and permanent cure in 83 per cent., and 3.5 per cent. of deaths. In the last 91 operations, since the above precautions

had been observed, he has not had a single death! C. H. Mayo had 9 deaths in 176 cases, but only one in his last 75; and Professor Halsted's results at Johns Hopkins are equally favorable.

In cases of long standing the exophthalmos never disappears, for the depths of the orbit have become filled with fat which continues to push the eye forward after the contraction of Muller's muscle has subsided.

Hypertrophy of the heart and secondary myocardial changes also remain, perhaps some cardiac weakness, but these are greatly diminished when the continuous cardiac excitation is removed.

It must be admitted also that, as Tinker states, the operation requires more skill and practice than most surgical procedures, and the prognosis is therefore far better done by a man whose experience in this line is considerable than by a surgeon of even excellent local reputation.

As regards the indication for operation, Kocher believes that "distinct vascular symptoms (other than mere palpitation and tachycardia) should at once induce surgical treatment." Before these have set in, the palliative method may be used for a while, and many cases may be relieved thereby or subject only to occasional recurrences. Should the mental symptoms and tachycardia persist or become more severe, the physician should recommend operation while the patient's general condition is still good, and should not wait until she is a complete physical wreck before turning over the responsibility to the surgeon. The surgeon should be allowed to operate upon the early but chronic cases which do not improve under palliative treatment.

These rules apply as well to the cases of "formes frustes" as to the outspoken Basedow's disease. The persistence of psycho- and cardioneuroses gives the indications, whether all the cardinal features are pronounced or not, and spontaneous recovery is no more likely to occur after the "forme fruste" has persisted than in cases where all the signs are well marked.

Sympathectomy (Jonnesco's Operation). — Another operation, which has been performed by Jonnesco, is the removal of the sympathetic ganglia on both sides of the neck. The result of this is usually an immediate slowing of the pulse, and often a cessation of other symptoms. Jonnesco reports several cases of permanent cure, but in the hands of a considerable number of later observers, among them Kocher, good results have been lacking or transitory, and this method should therefore be cast aside.

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III.

MISCELLANEOUS DISTURBANCES OF CARDIAC FUNCTION—THE SO-CALLED "CARDIAC NEUROSES" AND "CARDIAC NEURASTHENIA."

GENERAL CHARACTERISTICS.

One of the largest groups of cases seen by the clinician is made up chiefly of pale, anæmic-looking young patients, with hollow lustreless eyes and sunken cheeks, who complain of symptoms which may be divided into two categories:

Symptoms. — 1. Symptoms referable to sensory disturbances about the heart:—palpitation, precordial tenderness, pain or constriction, pains and sensory disturbances down the arms, and, in rarer cases, of attacks resembling angina pectoris.

2. Symptoms referable to motor disturbances of the circulation, and especially to the distribution of blood in the body:—cardiac arrhythmia, weakness, lassitude and weariness, vertigo, muscæ volitantes, fainting spells, and an infinite variety of psychasthenic and nervous symptoms.

This same symptom complex has already been encountered in the attacks of paroxysmal tachycardia (Chapter II.), where it has been seen to result from "arterial anæmia," or the relative depletion of the arteries through dilatation of the veins, especially in the splanchnic region. Y. Henderson believes that under these conditions the viens are not overfilled, but that they too have become depleted by transudation of fluid into the lymph and tissue spaces. Mr. C. C. Cody, in the Johns Hopkins medical clinic, has found a very low venous pressure (−2 to 7 cm. H_2O) in a number of cases of neurasthenia and post-operative asthenia in which the above-mentioned symptoms were present. The arterial pressure in all but one of these cases was about normal, ranging from 104 to 125 mm. Hg. This same circulatory state seems to be present throughout the groups of cases about to be discussed, although the mechanisms by which it is brought about are various.

Changes in Rhythm.—Alterations of rhythm are very common in this group of cases (Hoffmann, Mackenzie, Reissner). They are usually associated with respiration, with a slowing of the pulse during inspiration and a quickening during expiration (Fig. 325). It will be noted that this exactly corresponds to the normal centripetal action currents in the vagus (Fanthoven, Flohil and Battaerd) which occur with each inspiration, and it is probable that in the condition of heightened excitability this (usually subnormal) reflex stimulation becomes active. Stadler and Hirsch have been able to produce such irregularities by inflating the intestines of dogs and rabbits, but find them only accompanying dyspnœa. These observers also found that such inflations of the intestines were always accompanied by rise

of blood-pressure. The writer has been able to confirm these observations. Moreover, McCaskey and Russell find that an elevation of 30 or 40 mm. Hg in blood-pressure may occur in the course of gastro-intestinal troubles, especially hyperchlorhydria and flatulence. Russell suggests that there is a relationship between chronic gastric intestinal disturbances and sclerosis of mesenteric vessels.

In the cases of enteroptosis and of bathycardia a true pulsus paradoxus (diminution or dropping of beats during inspiration) may occur from the tugging upon the mediastinum, aorta, and great veins when the diaphragm is drawn down during inspiration. In rarer cases, and especially those of gastro-intestinal origin, small, early beats resembling extrasystoles are present. In making the diagnosis of extrasystoles, however, great care must be used, for it must be remembered that in the

FIG. 325. Respiratory arrhythmia in a young cigarette smoker

usual rhythmic variations in rhythm the last beat of a series with increasing rapidity may be followed by the pause due to maximal slowing, and thus an extrasystole may be simulated. On the other hand, it must be remembered that no experimenters have as yet been able to produce extrasystoles by stimulating the extrinsic cardiac nerves (Hering, Hoffmann, the writer and others), and therefore each case of the sort should be carefully studied with venous tracings and electrocardiograms. True ventricular extrasystoles demonstrable with the electrocardiogram are often brought on by flatulence, though many writers agree with Friedrich Müller that the presence of definite extrasystoles is indicative of myocardial disease.

Some years ago the writer had under observation a man forty years of age who was subject to palpitation and an arrhythmia brought on whenever he developed gas in the stomach or intestines. He himself was able to distinguish 'large and small' beats among the palpitations and tracings with mounted Erlanger apparatus from veins and arteries bore out his impressions. It is, however, difficult to decide whether these small beats represent auricular extrasystoles or whether there is simply a rapid rhythm interrupted by long pauses of vagal origin. He stated that nevertheless he was able to "outwalk his doctors" at hill climbing during periods when he was suffering from both palpitation and arrhythmia. A moderate dose (1 mg. = gr. $\frac{1}{60}$) of atropine caused dryness of the mouth and some vertigo, but did not greatly alter the pulse-rate nor cause the arrhythmia to disappear. The patient would not allow a larger dose to be given. If one could be certain that this dose had paralyzed the vagus the extrasystolic nature of the arrhythmia would be established, but it is most probable that the vagus was but little affected and that this evidence cannot be regarded as conclusive.

It has long been customary to designate such cases as "cardiac neuroses," or, from the neurasthenic symptoms which are most striking to the physician, as "cardiac neurasthenia." On closer examination, however, it may usually be found that both the cardiac and the neurasthenic symptoms are not primary, but

are secondary to some visceral displacement or irritation, to some intoxication, or in rarer cases to some primary intense emotional disturbance. The heart itself is sound, but, owing to the distribution of blood, does not get a chance to do the work of which it would be capable. The terms "cardiac" and "neurasthenia" are therefore both misleading, and it might perhaps be more satisfactory to designate such conditions by the adjective "pseudocardiac" ("pseudocardiac enteroptosis," "pseudocardiac gastralgia," "pseudocardiac aerophagia," etc.).

CLINICAL GROUPINGS.

Most if not all of these "cardiac neurasthenias" are brought on by the following conditions:

Alterations of the position of the heart in the thorax.
- *a.* Kyphoscoliosis, narrowness or flatness of chest.
- *b.* Cardioptosis or bathycardia (low heart),
  - (1) due to enteroptosis (low diaphragm),
  - (2) due to long thorax with diaphragm normal.
- *c.* High diaphragm from
  - (1) flatus,
  - (2) fat,
  - (3) tight lacing.

Although many cases arise in which no site of origin can be found for the symptoms, the following represent a few of the more common causes:

1. Abnormal position of the heart,
   - *a.* From curvature of the spine.
   - *b.* From pleural adhesions.
   - *c.* Owing to a low diaphragm.
2. Visceral reflexes.
   - *a.* Gastric, œsophageal and intestinal.
     - (1) Air swallowing.
     - (2) Gastritis, gastralgias.
   - *b.* Sexual organs.
     - (1) Sexual excesses (male or female).

       Female—At onset of menses and at menopause; at menstruation; from myoma and other lesions of generative organs.

       Males—Gonorrhœa, prostatitis, masturbation.
3. Intoxications.
   - Tobacco.
   - Alcohol.
   - Coffee.
4. Anaemia.
5. Intense emotional disturbances.

ALTERATIONS IN POSITION OF THE HEART.

DISPLACEMENT OF THE HEART FROM MALFORMATIONS.

The displacement of the heart which occurs in kyphosis and scoliosis is often the cause of a true cardiac weakness, *i.e.*, weakness and dyspnœa on exertion as well as from nervous causes—a so-called constitutional heart weakness as Kraus terms it.

The patients are usually pale, rather weak, and readily become exhausted and cyanotic, and manifest all the cardioneurotic symptoms. The actual cause of the trouble lies not so much in the heart as in the position in which it is placed in the thorax. Pressure and tractions upon both the venæ cavæ and the arteries render both inflow and outflow difficult, and thus bring about a high venous and a low arterial pressure, with the symptoms which follow in its wake.

Fig. 328.—Cross section of the thorax of a flat-chested individual, showing the systolic heaving of the chest wall (broken lines) and the forces bringing it about. The outlines of the chest wall and heart during the systolic heaving are shown by the dotted lines and obliquely shaded areas. The protrusions and retractions are shown by the arrows.

Kraus and recently Herz have called attention to the cardioneurotic symptoms which occur in all narrow-chested individuals. Herz calls attention to the fact that in such cases there is a tremendous lifting of the ribs and precordium with each systolic erection of the heart. This is due to the short anteroposterior and especially oblique diameter of the chest, so that the heart pivoted against the posterior chest wall must push out the left anterior wall in order to complete its systole (cf. Fig. 326). As will be noted, this condition is quite different from that which results from the low diaphragm or from cardioptosis, for in those conditions the heart either beats in the long axis of the thorax, or from its mobility can adapt itself to a narrower chest.

LOW HEART.

Even when there are no malformations of the chest, conditions arise in which the position of the heart within the thoracic cavity is altered, and these give rise to cardioneurotic symptoms. These conditions are:

I. Cardioptosis (wandering or movable heart), in which the mediastinal attachments are loose and the heart readily moves from side to side, as well as up and down.

II. Bathycardia (low or unsupported heart), in which the heart lies low in the thorax because the dome of the diaphragm is lower than normal. This is sometimes due to hepatoptosis and sometimes to a congenitally low liver.

III. The high heart (high diaphragm), from various causes, especially flatulence, fat, lacing, and during pregnancy.

MOVABLE HEART (CARDIOPTOSIS).

Cardioptosis, or extreme mobility of the heart as shown on change of position, was first described by Glenard (1885) and by Cherchevsky (1887). The latter observer noted that, while the borders of the normal heart move 1–3 cm. when the patient turns from the left side to the right (while lying down), a certain number of cases (2·4 per cent. of all cases, according to Einhorn) are encountered in which it moves from 4–7 cm.

without any other changes or any enlargement of the heart. As a rule, the symptoms date from some time when the patient has lost in weight, perhaps because the disappearance of mediastinal and omental fat causes the organs to become looser than before. Einhorn has found it much more common in men (18 cases) than in women (4 cases) and always associated with hepatoptosis; though in cases like that given below the element of hepatoptosis may be absent.

Case of Cardioptosis.

This, as well as the other symptoms, is beautifully illustrated by a case which the writer has recently seen in consultation with Dr. L. P. Hamburger. The patient, aged 31, had been a trained nurse since 18. Her father had died of enlarged heart and her mother died suddenly. Except for scarlet fever, whooping-cough, and measles as a child, she had been perfectly healthy until the age of 19, when a dermoid cyst of the left ovary caused profuse menstrual bleedings. This was removed and the wound drained. Adhesions formed, causing headaches and backaches and finally a nervous break-down, so that a second operation was done nine years later to relieve the adhesions. At the time of this operation she lost 14 pounds and was very nervous, and during her early convalescence had a syncopal attack during which her hands and forearms became completely blanched. One year later the patient felt her first cardiac symptoms, suffering palpitation, and when lying down has a feeling "as though the heart were turning over" or "like a rubber bulb or sponge being squeezed out." She then feels sick and has a feeling of oppression in the chest.

Physical examination shows a fairly nourished young woman of good color; pupils equal and no signs of Basedow's disease. The left lobe of the thyroid is slightly hard, but that organ is rather small.

The thorax is quite well formed, not especially flat. Costal angle normal. Lungs clear. The heart is not enlarged and the sounds are clear. On turning from side to side, however, the heart moves 8 cm. The pulse is of good volume and shows a well-marked respiratory irregularity of the type described above, but no extrasystoles. The abdominal walls are soft but not especially lax; the liver does not descend when the patient stands, but the right kidney is palpable.

Bromides, nitroglycerin, and strophanthus have been without avail; tincture of belladonna has somewhat quieted her cardiac symptoms. The intensity of the symptoms seems to vary with her general condition. Upon being assured of the trivial nature of her complaint, her symptoms **immediately disappeared.**

Several months later she reported, however, that they reappeared from time to time during periods when she was fatigued or nervous. The presence or absence of symptoms was always quite independent of the mobility of the heart. In spite of her gain in weight and the improved condition under treatment, the cardiac borders moved at least 7 cm. during the periods when she was free from symptoms.

Treatment. — The treatment of cardioptosis presents a number of difficulties. As seen from the case cited above, the symptoms depend not only upon the actual mobility of the heart but also upon the general condition of the patient's nervous system. It is the latter which determines whether or not the afferent impulses from the heart shall reach the threshold of consciousness. Accordingly, the unpleasant cardiac sensations may be present only when the irritability of the nervous system is increased by fatigue, anæmia, or other affections; so that relief of the latter by general measures relieves the cardiac symptoms as well, without affecting their underlying cause. The mobility of the heart itself cannot be treated directly; but it is sometimes possible, by overfeeding, to cause a sufficient deposit of fat in the mediastinum and pericardium

to diminish the movements a little. Even when unsuccessful in this way, however, overfeeding often aids by improving the general condition and nervous tone.

GENERAL SPLANCHNOPTOSIS.

The mechanism which gives rise to the cardiac symptoms of splanchnoptosis (enteroptosis) has been investigated anatomically by Keith and clinically by Wenckebach. The latter found, by means of the X-ray (fluoroscope), that the most important effect of enteroptosis was to remove the support of the liver and stomach from beneath the diaphragm. The dome of the diaphragm was thus usually seen to be flattened and to be situated a good deal lower than normal.

Fig. 327.—The low, normal and high hearts (Semi-schematic). I, first rib, X, tenth rib, VII, VIII, spines of seventh and eighth thoracic vertebræ. The horizontal line represents the "xiphisternal line" passing through the sterno-xiphisternal articulation. The small white arrow represents traction upon the trachea. A, low heart, B normal heart. C high heart.

The normal summit of the dome in quiet expiration is just above the level of the fifth rib, and its horizontal shadow just obscures that of the tenth rib behind. Keith finds that this is normally about 1 cm. above the "xiphisternal line," a horizontal line representing the level of the sternoxiphoid articulation. The upper border of the fifth rib at the junction with the cartilage is just at this level. In enteroptosis Wenckebach is able to see the X-ray shadow of the origin of the tenth and often the eleventh rib above the dome of the diaphragm, and the latter lies well below the xiphisternal line.

The writer finds that for ordinary purposes the most convenient landmarks are the xiphisternal articulation and the tip of the spine of the eighth thoracic vertebra which is just above the upper border of the tenth rib. The xiphisternal articulation, the dome of the diaphragm, and the tip of the eighth thoracic spine are normally on a level. In enteroptosis and low diaphragm the ribs drop so that first two structures are below the eighth spine while with a high diaphragm the ribs are raised so that they are above it (Fig. 327).

Effect on Respiration.—The effect of this low position of the diaphragm is exerted both upon the respiration and the heart. The abdominal respiration, which is due to the descent of the liver, is much diminished. For when the dome of the diaphragm is flattened, shortening of the diaphragm does not push down the liver, but pulls upon the lower ribs in a horizontal or even upward direction. The effect of this pull upon the lower

ribs (Fig. 327, A) is to narrow the cross section of the thorax (Duchenne) at this level and to draw the epigastrium inward, and thus by diminishing the air capacity in this portion of the lungs to decrease greatly the effect of inspiration both in sucking air and in sucking blood into the thorax. This is the so-called "paradoxical type of respiration." Naturally, its effect is to diminish the intake of air and thus greatly to enhance the effect of any cardiac insufficiency.

Effect on Circulation.—On the other hand, the lessened up-and-down movement of the diaphragm, coupled with the relaxation of the abdominal walls, greatly diminishes the force-pump and suction-pump action by which the blood in the abdominal veins is forced onward to the thorax. There is, therefore, a tendency for the blood to stagnate in the abdominal viscera. The venous pressure becomes low. In consequence, as Henderson and the writer have shown, the filling of the heart is less complete and the systolic output is diminished.

FIG. 328.—Radiograph of a patient with dropping heart (battledore). After Brugsch and Schittenhelm. The cardiac shadow is separated from that of the diaphragm by a well-defined space.

Leonard Hill has shown that if a rabbit is supported in the erect posture with feet down and head down, the blood-pressure falls and cerebral anæmia sets in. If one presses on the animal's abdomen, the blood-pressure rises at once. Erlanger and Hooker found that when normal men were supported and kept motionless in the vertical posture, the blood-pressure fell (*e.g.*, maximum fell from 120 mm. to 103 mm., minimal from 92.5 mm. to 86 mm., pulse-pressure from 26.7 mm. to 17 mm.[1]), and in one case there was "pallor, yawning, a feeling of warmth, faintness, nausea," and threatened syncope. These are the symptoms of arterial anæmia common in patients with enteroptosis.

The low position of the diaphragm exerts another effect upon the heart. The diaphragmatic platform on which it rests drops away like a trap-door and leaves it suspended from the great vessels and vertebral column by the aorta, trachea, mediastinum, and fasciæ. The heart thus lies in the longitudinal axis of the body; and, in systole, the apex can be seen to rise and to pull down on the trachea instead of moving inward. As Osler and Wenckebach have shown, a tracheal tug may often be felt and this may lead to a mistaken diagnosis of aneurism. However, this error may be obviated when the enteroptosis is taken into consideration, and especially when the tug diminishes upon pressing the liver upward and inward.

Moreover, when the low diaphragm descends in inspiration it exerts further traction upon the mediastinum and thus upon the aorta as well as upon the great veins, thus bringing about an inspiratory diminution

[1] The fall in pulse-pressure denotes diminished systolic output.

or dropping of the pulse-beats (pulsus paradoxus), exactly like that occurring in pericarditis, which the appearance of the patient may suggest.

Physical Signs.—The upper border of cardiac dulness in these cases does not usually extend above the third rib. The total area and the area of the cardiac shadow are usually diminished and the area of flatness completely obliterated. The attachments of the heart have reverted to the embryonic type, and that organ is suspended by the elongated pericardiac ligament. The apex is usually inside the mammillary line. In extreme cases the right ventricle is seen to beat in the epigastrium; but this often signifies only a dilatation of that chamber. The sounds are usually clear, but either sound may be reduplicated. There is usually a soft systolic murmur of accidental type over the area of the right ventricle, or occasionally at the apex.

The abdomen is often flat, and usually shows marked linea albicantes. The disappearance of subcutaneous fat makes the walls flabby and the viscera are easy to palpate. The liver can almost always be felt when the patient is sitting or standing. The kidneys are usually palpable and movable. When the patient stands, the viscera gravitate to the hypogastrium, where a fulness is seen, giving the abdomen the profile of an interrogation point turned upside down (Fig.).

Pathogenesis. Enteroptosis is far more common in women than in men, owing to the stretching of the abdominal muscles and relaxation of the perineal floor in pregnancy and labor. Hence it occurs more frequently in women who have not remained in bed long enough during the puerperium. Nevertheless, it is also common in single women and in men whose abdominal muscles are atonic from lack of exercise, or in persons who from any cause have rapidly lost weight. The rapid disappearance of the intra-abdominal fat uncompensated by contraction of the abdominal muscles takes away the support from the liver and facilitates the occurrence of enteroptosis. Indeed this latter factor is often more important than the muscular element, and it is not uncommon to find most typical examples of enteroptosis in thin persons whose abdominal walls are not abnormally flaccid.

Tight lacing, as well as causing atony of the abdominal walls, causes the viscera to tug at their ligamentous moorings and finally to stretch them, and thus bring on an enteroptosis. However, while the corset is being worn it pushes the liver and diaphragm up, the pelvic organs down. The typical corset heart is the high heart and not the low heart (see page 327, C). It is only when the corset is taken off that the heart and abdominal organs drop.

Treatment.—The treatment follows from the mechanical conditions. It is all-important to push up the liver. Fr Glénard, who first described enteroptosis, showed that symptoms were relieved by merely pressing upward on the abdomen with the hand (just as in Leonard Hill's rabbit experiment), and hence one of the oldest forms of treatment is the tightly fitting abdominal binder. A specially made corset arranged so as to bring an upward pressure upon all the structures below the costal margin gives excellent results, especially when supplemented by pads over the kidneys.

Probably the best form is an adjustable air cushion resting upon an aluminum plate that is strapped to the abdomen (Wenckebach). For a time the attention of physicians had been directed to the individual organ, especially the kidneys, and these organs were sutured into place. But experience has shown that this only remedies a small part of the trouble and does not remove the real cause.

The only method of real physiological therapy is one which will at once give support to the viscera within the abdomen and also restore the tonic contraction of the abdominal wall. This can be accomplished by accumulation of fat and by exercise. The former procedure is the one to be attempted first. If the patient can be kept at absolute rest in bed and overfed with a diet containing about 3500 or 4000 calories in twenty-four hours, a good deal of fat may be accumulated in about six weeks. The principal addition to the diet should be olive oil (15 to 25 c.c. three times a day = about 500 calories per day or about 60 Gm. (2 oz.) of adipose tissue). This can be taken between meals, pure or flavored with a little lemon, sherry, brandy, etc., to suit the palate. It is most important that the patient's digestion should not be disturbed by it. Salads with dressing, thick soups, and cereals (especially with cream) should be given in as large quantities as the patient will take, and she should be encouraged to drink milk instead of water. Butter and cheese are also valuable additions to the diet when the patient can be made to take them in liberal amounts. Cakes, sweets, and even puddings fall into the same category, provided digestion is perfect. She should receive milk or cocoa between meals and before going to sleep at night. In short, every means should be adopted to overfeed the patient. On the other hand, it must be remembered that if her digestion be spoiled in the process, it will be impossible to secure a permanent gain in weight, so that the process must be begun gradually and the patient's appetite should be stimulated to keep pace with the diet.

In order that the fat should be deposited in the places where it will give the most support (*i.e.*, the retroperitoneal tissue and gastrohepatic omentum), the patient should be made to lie with a pillow under the small of the back for as many hours as possible.

The result of the rest and overfeeding treatment, supplemented by careful bandaging, is most gratifying. With the return of intra-abdominal fat the patients usually improve in health and spirits, symptoms subside, and the element of cardiac weakness may entirely disappear. The patient's confidence in herself (or himself) returns and the neurasthenia subsides.

Case of Enteroptosis.

The following case, under the writer's care in the Johns Hopkins Hospital Dispensary, illustrates the course of the condition and the excellent results obtainable by treatment.

Mrs. Agnes L., aged 31, first seen Feb. 19, 1909. Complains of loss of strength, weakness, and palpitation especially on exertion. She is nervous and readily exhausted. Has had no swelling of the feet.

Family history negative. Patient was always healthy but has had diphtheria and is subject to sore throat. Chlorosis at 16. Bowels constipated. Menstruation regular but painful. She has had two children but no miscarriages. Drinks coffee and tea in moderation.

Present trouble dates from birth of last child four years ago. She feels tired all the time and is subject to weakness and palpitation after exertion. She sleeps well, however. Her feet are never swollen. Two years ago she was treated by another physician for the same trouble, which was then diagnosed neurasthenia. She was overfed and made to lie down every day. Gained weight and improved somewhat, but has lost weight since then.

Physical examination shows a fairly nourished woman, tall and sparely built. Her eyes and cheeks are sunken, and expression is one of depression. Her color is pale, but the hæmoglobin is 90 per cent. Thyroid is not enlarged. No glandular enlargement. Thorax is long and flat and held in the position of expiration. Costal angle is very acute. There are a few râles at the left apex (which were not present on subsequent examinations). The upper border of cardiac dulness begins at the third rib and extends in the fifth interspace to the left mammillary line and in the fourth 30 cm. to the right of the midline. Owing to the form of the patient's chest, however, the fifth left interspace is situated at a lower level (referred to the spine) than is normal. The cervico-xiphoid distance is long. Heart sounds are clear and pulse is regular.

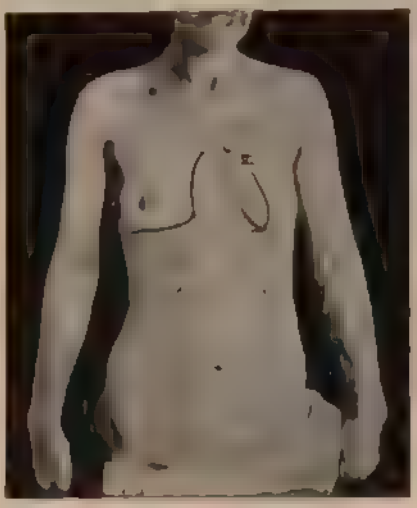

FIG. 329. Photograph of a patient with enteroptosis. The upper border of cardiac dulness begins at the third rib (III); the heart is small and lies vertically. The liver is low and palpable.

Abdomen. The liver extends below the costal margin. There is marked gastroptosis, the stomach lying below the umbilicus. Both kidneys palpable. Genitalia negative.

Extremities.—Sensation and reflexes normal.

Patient was given an abdominal bandage and encouraged to full diet, especially rich in butter, milk, eggs, and salad. Besides this one tablespoonful of olive oil three times a day. She was made to rest and lie down several hours a day with a pillow under the small of her back to favor deposition of perirenal fat.

Within an hour after the abdominal binder was first put on her condition was markedly improved, her expression was brighter, and she felt more active. The patient, however, still stood with stooping shoulders, which caused the chest to continue in the position of expiration and allowed the heart to hang low. This position was improved by the use of shoulder braces. The patient's condition and strength steadily improved and her cardiac symptoms had entirely disappeared after the abdominal bandage was put on. The gain in weight during three months was only 3½ pounds.

Case of Enteroptosis Simulating Aneurism of the Descending Aorta

P. R., a wool sorter, aged 42, came to the Johns Hopkins Hospital Dispensary on March 5, 1909, complaining of a drawing pain in both sides and in the epigastrium. The family history and personal history were negative. The patient denies lues, but has had to lift heavy sacks in his work. His pain began about six weeks before admission while he was at work and was accompanied by palpitation, and it has continued since then.

The patient was a well-nourished man of good color. The left pupil was larger than the right, but both reacted to light and accommodation. The thorax was long and there was a slight funnel breast. The lungs were clear on percussion and auscultation. The area of cardiac dulness was slightly smaller than normal; dulness began above at the lower border of the third rib, extended 7.5 cm. to the left in the fifth left interspace and 2.5 cm. to the right of the midline. The heart moved 6 cm. on change of position. The apex is 3 cm. below the xiphisternal line. The left radial

pulse was slightly smaller than the right. There was well-marked pulsation in the epigastrium, and the liver was well seen and readily felt below the costal margin. There was a well-marked tracheal tug which diminished when the liver was pushed upwards with the hand.

The fluoroscopic examination by Dr. Baetjer showed that the aorta was clear. An abdominal binder was applied. The abdominal pains and palpitation ceased and the tracheal tug diminished markedly. The patient was able to continue work without discomfort. His pains have been absent for over a year.

LOW HEART WITHOUT ENTEROPTOSIS (BATHYCARDIA).

There is another type of long, flat-chested individuals in whom, although there is no enteroptosis, the diaphragm is low. The dome of the diaphragm is not flat, but is well arched. The insertion of the diaphragm may be somewhat lower, and the length of the thoracic cage, which is held in the position of expiration, is considerably greater than normal (Fig. 327, A). As a result of this lengthening of the thorax, the distance from the structures upon which the heart hangs (aorta, trachea, mediastinum) to the diaphragm, which supports it, is lengthened, and just as in enteroptosis the heart hangs free above the diaphragm. It is therefore termed the "hanging heart" or "dropping heart." It pulls upon the trachea in systole and causes a tracheal tug. It pulls upon the aorta in inspiration and causes a pulsus paradoxus. The interference with cardiac filling and with the abdominal circulation gives rise to about the same symptoms of cardiac weakness as are encountered in enteroptosis, though often to a less marked degree.

The diagnosis is usually best made with the fluoroscope; for the presence of a mild brachial impulse and pulsus paradoxus may cause the condition to be mistaken for either aneurism or mediastinitis. There may even be a slight tugging on the low diaphragm (Broadbent's sign) at the depths of respiration. It is extremely difficult to exclude mediastinopericarditis in many cases in which the palpitation, pallor, fatigue, shortness of breath, paradoxical pulse, etc., are intense. In some cases with reduplicated first sound mitral stenosis may be thought of. Absence of hypertrophy of the left ventricle (cardiac dulness inside mammillary line) should exclude organic mitral insufficiency even in the presence of a systolic murmur. The diagnosis is made chiefly on fluoroscopic examination.

Treatment.—As the condition is due to the low diaphragm, just as in actual enteroptosis, the chief indication is to raise the diaphragm. In bringing this about with normal abdominal walls a bandage is of some avail, but fattening is not successful.

On the other hand, the obliquity of the ribs is also at fault, and this can be corrected by training the patient to take deep inspirations and to stand with his shoulders and hips thrown back.

HIGH DIAPHRAGM.

The exact opposite condition, that in which the diaphragm is so high that the heart is placed in a position in which it works at a disadvantage (probably by interference with venous inflow), is found in fat persons, in many dyspeptics with flatulence, in emphysema, and in women as a result

of tight lacing. In the first three conditions there is diminished respiratory movement, especially the costal movements, since the ribs in most cases are held in the position of expiration and the possible excursion thus diminished; while in persons who lace tightly abdominal respiration is impeded and the respiration is mainly costal. In these cases the heart is raised by the diaphragm, especially in inspiration, and thus comes to lie more transversely to the axis of the body. In such persons the xiphisternal articulation lies above the level of the eighth thoracic spine, the diaphragm shadow, according to Wenckebach, obscuring the ninth and tenth ribs. The apex lies in the fourth interspace o u t s i d e the mammillary line, often leading to the suspicion of valvular lesion or myocarditis.

This pushing up of the heart tends to impede the heart's action and to produce fall of arterial pressure, as was first shown by v. Frey and Krehl in 1890.

The clinical result of these conditions is to produce a syndrome not unlike that of the exactly opposite conditions, "cardioptosis" and "dropping heart," – *i.e.*, a diminished cardiac filling,—and is undoubtedly in a large measure responsible for many of the symptoms of the "heart of obesity" and of indigestion.

Treatment must be directed to the cause, – regulation of diet for the fat and dyspeptic, loosening of the corset for the woman who laces. However, the latter should be done gradually enough to give the abdominal walls time to adjust themselves, lest a true splanchnoptosis replace it.

REFLEX CARDIAC DISTURBANCES.

GASTRO-INTESTINAL.

Patients with chronic gastro-intestinal disturbances often come to consult the physician for the cardiac symptoms which these bring about, namely, palpitation, pain in the region of the heart, tachycardia, and often irregularity of the pulse,—symptoms which are all more deeply impressed on the patient's mind than are the heart-burn and belching from the underlying indigestion.

As has been seen in connection with angina pectoris, gastric disturbances may cause cardiac symptoms. The motor disturbances (arrhythmia, tachycardia, etc.) are in a large part due to the spread of stimuli from the gastric branches of the vagus to the cardiac, while the sensory symptoms are due in part to false reference of impressions, in part to a similar spreading of stimuli and in part to an associated hyperæsthesia of these branches of the vagus.

The chief irritants are butyric and lactic acids (acid fermentation), excess of hydrochloric acid, and the gases of fermentation,—CO_2 (40 per cent. in the absence of HCl), H_2, N_2, O_2, H_2S, and often CH_4 (inflammable) in butyric acid fermentation (Hoppe-Seyler).

Air Swallowing.—One of the most important factors in pseudocardiac dyspepsia is a i r s w a l l o w i n g. As Wylie has shown, it is extremely common for persons suffering from slight gastric discomfort to find themselves relieved by belching. As a result they seek further relief by forcing

themselves to belch. The forced belching gives only momentary relief, but aggravates the discomfort, giving rise to a familiar sensation of an object lying just behind the larynx. They belch again to remove it, and the belching is thus continued indefinitely, always accompanied by a certain discomfort and often by a loud noise.

Mechanism of Aerophagia.—Wyllie and others have shown that the mechanism of involuntary and voluntary belching is quite different. In the former case an excess of the gases of fermentation is regurgitated from the stomach, and thus can occur only when there is an excess of gas. In the latter case the patient first swallows or gulps the air by placing the tongue against the roof of the mouth (in the position of pronouncing the consonant "T" and then exerting a deep inspiration. These movements force the air into the œsophagus. It remains there an instant, and may then be either swallowed or expelled by a forced expiration with the glottis closed, causing the loud noise of belching as it forces apart the vocal cords and pushes up the epiglottis. Most often part of the air is swallowed and part regurgitated, and a few bubbles of air remain in the œsophagus most of the time, giving rise not only to the feeling of discomfort but often to reflex cardiac disturbances. Wyllie calls attention to the fact that air gulping occurs not only in man but also in horses and cattle, where the condition is known as "wind colic" and "hoven," which often becomes so severe that it may cause the death of the animal. The symptoms are "difficult breathing, bloodshot eyes, red mucous membrane, loud tumultuous heart-beat, trembling of front legs, etc." This can be brought about in dogs by inflating either stomach or intestines with air under pressure, paralysis and heart-failure resulting. Wyllie believes that the condition is still more common in infants and in children, and thinks that it is responsible not only for wind colic but for certain cases of death with abdominal distention.

Palpitation when patients are quiet may be more striking than actual shortness of breath on moderate exertion (unless anæmia is also present). But this is not an invariable rule, for on account of the high diaphragm of flatulence, the intensity of the cardiac discomfort, or, on the other hand, the habitual weakness of the patient's muscles, there may be actual cardiac weakness as well.

The treatment of air gulping is of the greatest importance. Wyllie states that this troublesome habit can be promptly overcome by keeping the mouth open. For persons who swallow air in their sleep, a gag or cork has to be tied in the mouth. This method is in general use among veterinary surgeons and is uniformly successful. It is evident, therefore, that the diagnosis of air swallowing must be carefully made. In many cases this may be done by getting the patient to show you how she usually belches, the voluntary procedure indicating the nature of the process. In doubtful cases it may be necessary to analyze the gas by Hoppe-Seyler's method; but, as Wyllie remarks, the diagnosis is best made ex juvantibus, by cessation of the condition when the mouth is kept open. The possibility of unconscious quiet air swallowing in other cases of flatulence must also be borne in mind.

Constipation.—An accumulation of fecal matter is also a very common cause of cardiac symptoms. Extrasystoles are usually more common when the patients are constipated. Kuthan has also seen patients in whom attacks of angina pectoris occurred regularly during periods of constipation and disappeared when the bowels were kept open. These symptoms are produced partly by the lifting of the diaphragm and perhaps also in part by the chemical action of indol, skatol (Russell, Herter), and other fermentation products. These substances may act directly upon the intestinal nerve endings or upon the heart muscle and

cardiac nerves after absorption into the general circulation (as assumed by McCaskey). It must be admitted that the action of such products is not very well-known.

Apart from the general methods, treatment of the cardiac symptoms is best accomplished by treating the gastric condition with appropriate diet, lavage, galvanization (8 to 12 milliamperes), and faradization of the stomach, etc. The bowels should be kept open. The various forms of fermented milk containing lactic acid (buttermilk, kephyr, etc.) as well as the preparations of lactic acid bacilli are often of benefit in the treatment of intestinal fermentation.

Treatment of the constipation which is often present should consist of free purgation with Epsom salts, followed by a course in cascara, hydrotherapy, abdominal exercise, and a diet rich in fat, coarse foods, and in liquids. The most important feature is forcing the patient to defecate at regular hours and at no other times.

REFLEXES FROM THE SEXUAL ORGANS

Practically all the disturbances of the sexual organs are accompanied by the pseudocardiac syndrome. It occurs in both male and female after sexual excesses and organic diseases.

MALE SEXUAL ORGANS.

In men gonorrhœa, prostatitis, and especially masturbation are among the first conditions to be thought of when a patient presents himself with these symptoms. Curschmann and Bachus have especially called attention to the latter condition and have found that masturbation may even lead to cardiac hypertrophy. Bachus has made the very significant observation that in many of his masturbators the thyroid glands were somewhat enlarged, so that he believed that the abnormal sexual activity might have led to a secondary over-activity of the thyroid. (Perhaps this may occur through the action of a hormone in the testicles, seminal vesicles, or prostate.) At all events, since this organ may be affected reflexly, the secondary activity of the thyroid is to be thought of in all cases of pseudocardiac disease of sexual or of purely nervous origin. It is also possible that the prostate gland may have an internal secretion of similar character.

The diagnosis must be made from the history, as well as from a careful physical examination in which the mouth of the urethra is especially examined, and careful palpation of the prostate and prostatic secretion.

Treatment is directed mainly to the primary condition, but the bromides should be used somewhat more freely in sexual disturbances than in the other conditions.

FEMALE.

In women the pseudocardiac disturbances are not only more common but more severe than is usual in men. Palpitation, with tachycardia and weakness, is very common at the age of puberty and is practically universal in chlorosis which is then so common.

In young and healthy married women Kisch has found that attacks of palpitation with rapid pulse and dyspnœa may occur—

1. As the result of sexual excesses in women whose sexual desire is very keen.
2. After attempted coitus when vaginismus is present (cured by operation upon the vagina).
3. In women who have practised coitus interruptus for a long time without attaining sexual satisfaction.

Kisch believes that in general coitus stimulates the cardiac nerves in proportion to the intensity of the orgasm. Masturbation, on the other hand, has a much less intense effect on women than on men (Kelly), and is rarely responsible for cardiac disturbances.

Veit calls attention to the fact that asthmatic attacks from cardiac insufficiency constitute an early symptom of myoma. He believes that these arise in the early stages of brown atrophy and cardiac obesity, as these lesions have been found by Lehmann and Strassman and also by Fleck in 40 per cent. of all cases of myoma.[1] Kelly and Cullen, however, deny that myomata in themselves are associated with any cardiac disturbances except those due to the anaemia which is present. In a very careful study of 1428 cases of myoma they found that "In the majority of cases (with cardiac manifestations, 92) an apical systolic murmur was detected. This murmur was usually very soft in character. In some it was limited to the apex, but in others it could be traced to the axilla and in some patients to the base of the heart." In nearly all the cases (92) in which cardiac lesions were present, the patient gave a history of menorrhagia, often associated with intermenstrual bleeding. These patients, as a rule, stood the anaesthetic well and in a comparatively short time they had gained much in strength and their cardiac murmurs had disappeared. Some authorities claim that the myoma in itself brings about cardiac changes. If such were the case, then the larger the myoma the more pronounced should be the cardiac murmurs. This has not been our experience. The largest tremors have not been associated with any cardiac symptoms, but the heart complications have almost invariably been associated with copious bleeding from the uterus (*i.e.*, submucous myomata).

"Most of the murmurs noted in our cases were at the time considered to be functional." (Cullen.) That there is little danger from functional impairment of the heart is shown by the fact that Kelly and Cullen's mortality in their last 240 cases of myoma was less than 1 per cent.

Lenhartz has found that cardiac symptoms are almost always more frequent during the week preceding the menstrual flow. They are also more frequent as the climacteric is approached, an angioneurosis (flushing) with palpitation and more or less tachycardia being universal. Strassman and Lehmann have called attention to the similarity between these phenomena in ovarian secretion and those of the thyroid gland, and, as has been stated, the thyroid varies in size with the changes in ovarian activity. Leo Loeb, Starling, and others have shown that this is due

[1] In 34.6 per cent. of Fleck's cases there was no anæmia.

to the action of a hormone arising in the ovaries and especially in the corpus luteum, which acts upon the uterus, mammary glands, and thyroid. No doubt it also has some action upon the heart, but this is still obscure, and it is difficult to determine how much of the effect is due to the ovarian secretion itself and how much to the secondary hyperthyroidism.

Prognosis and Therapy. The prognostic importance of cardiac disturbances arising in the sexual organs varies with the primary lesion and its chronicity. In the presence of gonorrhœa or pelvic abscesses the probability of a metastatic myo- or endocarditis must not be forgotten. In the presence of anæmia the development of fatty degeneration and even of insidious mitral stenosis must be borne in mind, while in the presence of myoma myocardial changes which vary from primary hypertrophy to a brown atrophy and cardiosclerosis (due chiefly to the anæmia) must be thought of. In masturbating men there is a true cardiac hypertrophy with the usual accompanying changes. These factors must, therefore, be excluded before the diagnosis of a true neurosis is made and an unqualified favorable prognosis can be given.

In the simple cardioneurosis or pseudocardiac sexual disturbance the cardiac outlook is favorable if the primary condition can be removed. If not the prolonged reflex stimulation of the cardiac nerves leads first to a "work hypertrophy" and then probably to cardiac overstrain and premature cardiosclerotic or atrophic changes.

The treatment is therefore in the field of the gynæcologist or genitourinary specialist and not in that of the internist. Even masturbation and sexual excesses may have a basis in organic irritation and should not be regarded as entirely psychogenic without examination. These may be much helped by psychotherapy, cold baths in the morning and cold packs at night, and exercise during the day. The psychic effect of the treatment will be greatly enhanced if the impression is clinched at once by the administration of potassium or sodium bromide (1 Gm.—gr. xv, t.i.d. and before going to bed) disguised in elixir of calisaya or in compound tincture of gentian or of cardamom; for the patient's confidence in the outcome is gained by finding the abnormal desire to decrease at once with the onset of treatment.

ADENOIDS AND RESPIRATORY OBSTRUCTION.

Adenoids.—The presence of adenoid growths in the nasopharynx is also of importance, not only because they interfere with the respiratory intake of air and thus bring about dyspnœa on exertion, which may simulate a true cardiac weakness, but also because attacks of mild asphyxia may occur during sleep and cause the patient to awaken suddenly with a severe palpitation and other cardioneurotic symptoms. Besides this, during waking hours the lesions may continue to produce reflex irritation of the cardiac nerves and give rise to cardioneurotic symptoms in the same way as do disturbances in other organs.

Arrhythmia of Nasal Origin.—A physiological basis for these clinical findings has been furnished by François-Franck (1889), who found that an arrhythmia of vagal origin could be produced by stimulating the nasal

mucosa. His studies have recently been confirmed by Koblanck and Roeder, who found that in 8 cases with arrhythmia and nasal disease there were alterations in the mucous membrane of the nasal septum in a spot opposite the middle turbinate bone. There were often nose-bleeds as well. Touching this spot with a blunt probe in man and animals produced a similar arrhythmia. No other area of nasal mucosa gave this reflex. The arrhythmia was characterized by series of beats with increasing rapidity interrupted by long pauses, sometimes simulating extrasystolic bigemini (Curves 1 (man) and 3 (rabbit), K. and R.), but it could not be produced after either vagus was cut. Stimulation of other mucous membranes in this manner did not give rise to such arrhythmia. The authors showed that these stimuli are carried by the septal branch of the trigeminus which lies in this vicinity, for they could not be produced after cutting the trigemini, and believe that they are carried directly from the trigeminus nucleus to that of the vagus through the fasciculus longitudinalis medialis. With cure of the nasal condition the arrhythmia and allied disturbances disappeared.

Cardiac Asthma from Disease of Nasal Septum.—François-Franck also showed by careful graphic methods that stimulation of the nasal reflex can give rise to cough, laryngeal spasm, asthma, and even a reflex bronchitis, reflexes which in themselves may add to the impression of a primary cardiac disturbance. He found that these reflex conditions were more pronounced in animals with aortic insufficiency than in normal animals. The condition in man is similar, and in the presence of an organic cardiac lesion these contributing factors may play a rôle which determines the security of the cardiorespiratory symptoms, so that the cause of the paroxysmal dyspnœa may in some cases have to be looked for in the nose.

TOXIC CARDIONEUROSES.

"TOBACCO HEART."

Persons who suffer from excessive use of tobacco may be divided into three classes:

1. Non-smokers suffering from a single indulgence (acute tabagism or nicotinism).
2. Young habitual smokers, especially those who inhale cigarette smoke (subacute nicotinism).
3. Old habitual smokers, especially of cigars and pipes, who suffer from the pathological changes produced in the arteries (especially the coronary arteries) and myocardium, and partly from the added effects of the nicotine.

PHYSIOLOGICAL EFFECTS.

The physiological effect of smoking has recently been studied by Lee, as well as by Bruce, Miller, and Hooker.

Lee found that ordinary tobacco smoke obtained from 1000 Gm. tobacco contained nicotine 1.165 Gm., pyridine bases (chiefly pyridine and collidine) 0.148 Gm., HCN 0.08 Gm., NH_3 0.36 Gm., CO 410 c.c. The chief toxic product is therefore nicotine. The composition varied considerably with both the quality of the tobacco and the mode of smoking. The greater part of the nicotine at the seat of combustion is destroyed, and that

which reaches the mouth is volatilized by the hot gases while passing over the unburned area. Accordingly a thick cigar has the worst effect, since it acts as a chimney leading the gases to the mouth, while in a thin cigar, "stogie," or cigarette they escape into the surrounding air. (In cigarette smoking inhaling the smoke more than compensates for this difference in combustion.) In long-stemmed pipes much of the nicotine condenses before reaching the mouth.

Lee found that in non-smokers the first effect of smoking a cigar was to produce a rise of 10–20 mm. Hg in the maximal blood-pressure, which was often associated with palpitation. Within five minutes after this the maximal blood-pressure fell 50 mm. Hg, and this fall was accompanied by pallor, sweating, weakness, and colicky pains in the abdomen, as well as by the appearance of muscæ volitantes, irregularity and weakness of the pulse,—or, in other words, the symptom complex of arterial anæmia.

In more habitual smokers, those of the second group, a single cigar produced only the rise of blood-pressure and palpitation. The subacute symptoms, therefore, come on only as the result of excessive indulgence.

In old habitual smokers these observers found either no effect whatever or only a slight rise of pressure resulting from a strong cigar, without any of the disagreeable symptoms.

Lee's observations have been repeated by Bruce, Miller, and Hooker, who found that smoking increases the maximal, minimal, and pulse-pressures in man, though later these return to normal. The cardiac output, therefore, seems to be increased at first, as Lee had found in cats. Bruce, Miller, and Hooker also found that the volume of the hand always decreased during smoking (vasoconstriction), whereas Lee found that the volume of the cat's intestine also decreased. It is probable that a little later there occurs, in man, a dilatation of the abdominal vessels, but it is not yet certain that it does so.

The chief sufferers from tobacco are the young cigarette smokers who inhale the smoke and thus soon suffer immediately from the physiological effects of the nicotine. They complain of weakness, giddiness, intense palpitation and tachycardia (from continued stimulation of the cervical ganglion cells), and often of irregularity of the heart, which may be very distressing. It is most noticeable that the intense sensory disturbances occur without any motor insufficiency of the heart. Thus, a young man of 20 years, an habitual inhaler of cigarette smoke, recently consulted the writer, complaining of fatigue, giddiness, muscæ volitantes, intense palpitation, but, on further questioning, stated that he was in the habit of running a quarter of a mile every evening for exercise, and after this exercise he had neither palpitation nor shortness of breath! Needless to say, he improved at once after stopping tobacco.

On the other hand, all sufferers from nicotine are not free from motor symptoms nor do they recover so readily. In many cases the nicotinism is supplemented by the use of alcohol, and secondary myocardial changes, and in the older persons arteriosclerotic changes, have been superinduced.

In the middle-aged smokers the symptoms are chiefly those of angina pectoris and precordial pain. Very commonly this is a true angina of coronary sclerosis, but there is a certain number of cases in which the unpleasant symptoms completely subside upon cessation of smoking.

It would be a very fascinating hypothesis to believe that in such cases the effect of smoking is to produce a transitory constriction of the coronary arteries and this to cause the symptoms, but, on the contrary, some recent experiments upon dogs, done under the writer's direction by Dr. George Bond, have shown that the flow through the coronary veins is actually increased by smoking.[1] It is probable, therefore, that in early tobacco poisoning at least, the sensory symptoms are due to stimulation of the cardiac nerves and not to ischæmia of the myocardium. The commonness and insidiousness of coronary sclerosis, however, render it difficult to decide in any individual case whether the effect is entirely functional or has also a basis in arterial changes.

COFFEE AND TEA.

The palpitation, tachycardia, and tremor which result from over-indulgence in coffee and tea are familiar to most persons from personal experience. They often manifest themselves in chronic forms and cause cardioneurotic symptoms. Precordial pain and tenderness are quite common. Foote and Simpson, under D. R. Hooker's direction, have found that when a person accustomed to coffee takes a cup of it there is a transitory rise in maximal and minimal blood-pressure and a slight vasoconstriction of the hand. In persons unaccustomed to coffee these changes are much more intense. Indeed this partial immunity to coffee is very transitory, for the writer has found that after discontinuing its use for several months a single cupful would give rise to palpitation, tachycardia, and insomnia, while a few months before and a few months later two cups could be taken at a time without producing symptoms.

Coffee, like tobacco, gives rise to sensory cardiac symptoms by increasing the irritability of the nerves without causing any motor insufficiency, and consequently the patients, as a rule, do not show muscular or cardiac fatigue on exertion in spite of the symptoms.

Tea.—Owing to its content of caffeine, tea causes the same symptoms as coffee, but is less extensively used in large quantities. In England, however, similar cases are occasionally reported.

ALCOHOL.

Palpitation and the other symptoms of "cardioneurotic" (pseudo-cardiac) weakness also occur in persons who take alcohol in quantities that are just in excess of their tolerance, and the possibility of this cause must be borne in mind. In some individuals, as in Reissner's case, palpitation and irregularity may follow the ingestion of a single glass of wine, without any symptoms of intoxication setting in. That these conditions may continue without the patient's recognizing the cause is a common experience, and a considerable number of cardioneurotic cases result from this unintentional over-indulgence in alcohol. Women and young persons are more sensitive than men. The functional power and endurance of the heart muscle is, moreover, weakened by alcohol; and acute dilatation may set in from comparatively slight exertion. If the use of alcohol is long continued, it may lead to fatty and fibrinous myocardial change, but this in mild cases subsides when the cause is removed.

[1] Bond registered the outflow from the coronary veins by the drop method.

SIMPLE EMOTIONAL CARDIONEUROSES.

As has been seen, by far the greatest number of so-called cardioneurotic cases are of postural, reflex, or toxic origin. However, it still remains beyond question that emotional disturbances alone, or in conjunction with other conditions which in themselves are not sufficiently intense to produce symptoms, may give rise to cardioneurotic symptoms. Palpitation and even precordial pain are almost universal after severe emotional disturbances and shocks and during periods of worry.

The motor effects are usually shown by tachycardia, though occasionally arrhythmias may occur. This the writer has observed upon himself on an occasion of intense emotion, during which the pulse became extremely rapid and seemed either to drop an occasional beat or to give rise to an extrasystole. When the cause of the worry was removed, within five minutes the pulse again became regular, so that the arrhythmia could not be accurately studied nor has it recurred at any other time during the four years that have elapsed.

Similar cases are found in the literature (Reissner). In rare cases an emotional shock may cause death, even when the heart is otherwise healthy (Gibson), but the nervous mechanism by which this is brought about is not clearly understood. It is probably a condition of exaggerated vasomotor shock arising in response to a cortical stimulus, just as it may result from over-stimulation of a peripheral nerve. In most cases of the sort, however, the heart and especially the coronary arteries are already diseased (see page 281).

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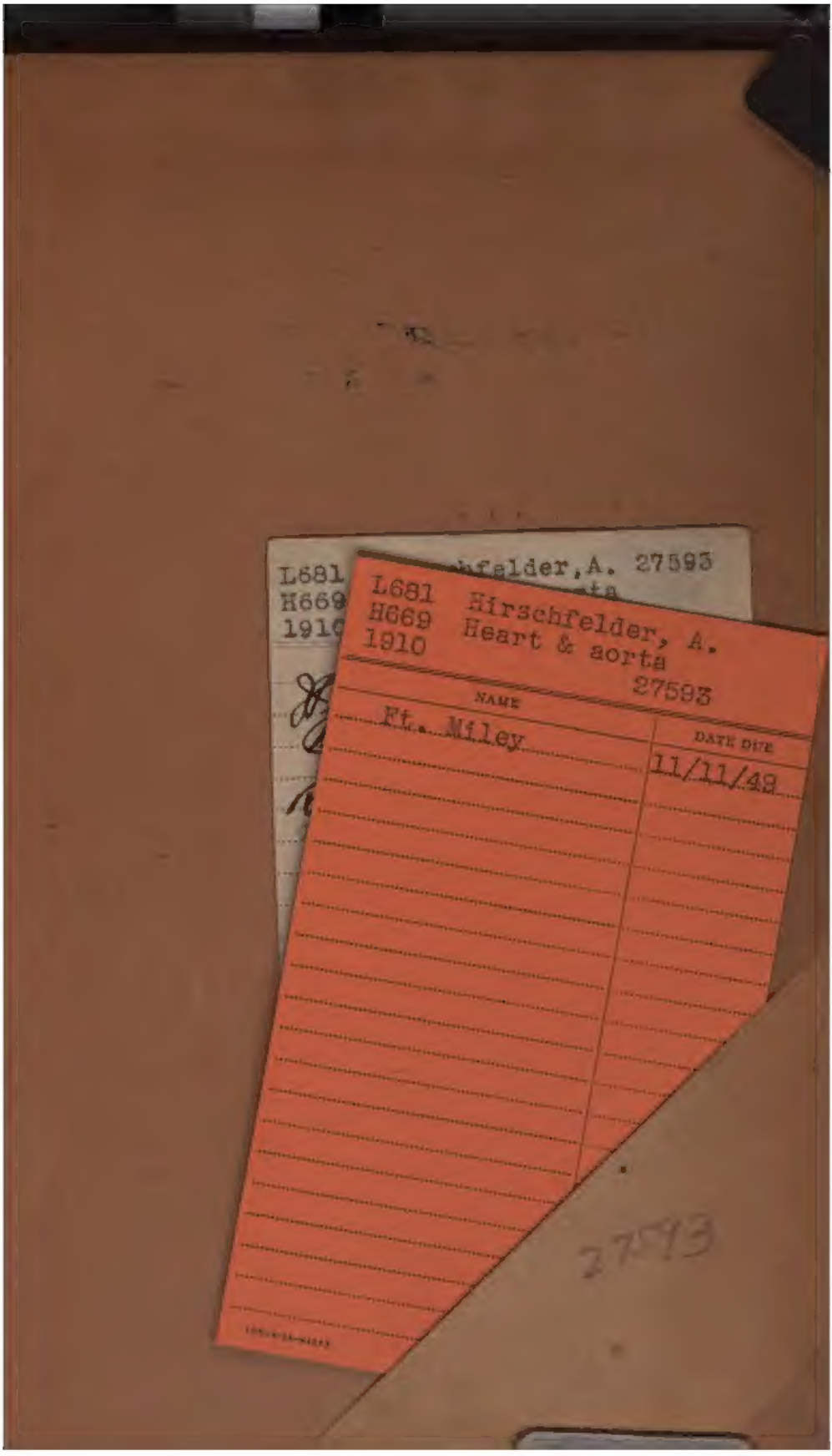
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